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NEUROLOGICAL BULLETIN

CLINICAL STUDIES OF NERVOUS AND MENTAL
DISEASES IN THE NEUROLOGICAL DEPARTMENT
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EDITED BY FREDERICK TILNEY, M.D. PH.D.

VOLUME II
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NEUROLOGICAL BULLETIN

VOLUME II

JANUARY, 1919

NUMBER I

CHRONIC CHOREA WITH A DISCUSSION OF TREATMENT

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NEW YORK CITY

The patient, C. W. (11871 and 21309), is a girl 16 years of age. She presents a somewhat emaciated, stunted appearance and has a listless manner.

PRESENT ILLNESS. *Complaint.*—She first appeared at the Clinic September 5, 1917, with symptoms as given below, and was diagnosed as a chorea. She came again May 11, 1918, having been referred by the Roosevelt Hospital and also, apparently, by Social Service.

Some two years before the latter date she had an attack of nervousness, her hands and lips shook and she dropped things. This lasted several months. A second, similar attack began in the summer of 1918 and lasted about two months. Before each attack she had vomiting and abscesses and was "run down." She "vomited for three months, couldn't keep things on her stomach" during both attacks. Blood was vomited while in the hospital. She had fever, especially in the afternoon. Besides the above she had a bladder trouble which became worse during these attacks. The bladder trouble dated from childhood and was concealed from her mother. She used to have nocturnal enuresis and also occasionally diurnal, in school. Her later bladder trouble, however, which was worse during her nervous attacks, was a difficulty in micturition, for which she had to be catheterized. She had been attending Roosevelt Hospital for five months previous to May, 1918, apparently on account of her bladder trouble. Blood was also passed, its exact origin not having been determined. A catheterization performed during November,

1918, gave a considerable quantity of bloody urine, not presenting the appearance of fresh blood.

She complained of constipation but also apparently had had some involuntary defecation, the first about four years ago. She also complains of a feeling of heaviness and numbness in the legs during her attacks of nervousness and of a shortness of breath—a tight feeling produced by going upstairs and by drinking much water. She is obliged to sleep with three or four pillows under her head. Her appetite is poor. She has slight headache at times and has a general feeling of fatigue. Has no pains except those connected with her bladder trouble. She has always had amenorrhœa. She had mumps when four or five years old. She has had measles and pertussis and had "rheumatic fever" at ten years of age. She was operated on for appendicitis in June, 1916, and was "nervous" already at that time.

FAMILY HISTORY.—Her father died in April, 1918, of some intestinal trouble (cancer?). Her mother is alive and well. She has four brothers and two sisters living, and healthy except one brother who was in the hospital for ulcers of stomach (or appendicitis?). Two children died, one at 1½ years (convulsions?), the other by accident.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—The patient has a kyphosis.

COORDINATION.—This is normal in the usual tests, except that while they are performed choreiform movements are present.

Abnormal Involuntary Movements.—Choreiform movements are present in fingers, arms, trunk and legs.

Reflexes.—The biceps, triceps, patellar and Achilles are all active. There was no clonus. The abdominals are difficult to elicit but are apparently, in part at least, present. There seemed at times to be a doubtful Babinski, but this appearance was probably due to the choreiform movements.

Muscle Strength.—There appeared to be no real paresis, but an intermitting in opposing passive movements, such as is seen in chorea. These intermissions in tension were especially marked in extension of the feet and thighs and adduction of the thighs.

GENERAL SENSORY. *Touch.*—Touch acuity was normal except a possible slight diminution on the feet. Touch localization was normal on the upper extremities (lower not tested). Touch discrimination on the second finger tips was 3 to 4 mm.

Temperature.—This was normal excepting a slight diminution on the feet (and legs?). The fact that the feet were rather cold at the time may account for this.

Vibratory.—This was apparently normal on knuckles and elbows but either diminished or absent on ankles, shins and big toes. (The patient "feels it different but not a buzzy feeling like the upper extremities".)

Muscle-tendon.—Flexion and extension of the big toes and of the foot are defective, apparently being always, or nearly always, felt in the wrong direction. Abduction and adduction of the feet are felt normally. Movements of fingers, hands and arms are correctly felt, but the patient is slow in putting, with eyes closed, the corresponding part in positions corresponding to those of the part passively moved.

The above was the result of the sensory examination in May, 1918. Another examination on November 4, 1918, gave the following results:

Touch.—Acuity and discrimination were not quite as good on the right as on the left. There was no astereognosis.

Pain.—This was apparently slightly better (sharper) on the left foot and leg than on the

right; a similar difference in arms and face was much more doubtful.

Temperature.—This also seemed better on the left side.

Vibratory.—This was now normal on shins and ankles, with possibly some diminution on the big toes.

Muscle-tendon.—Appreciation of movements of left big toe, ankle, knee, arm and hand was normal, but there was some uncertainty on the right.

The patient said the nervousness (chorea) was worse in the right arm and that she dragged the right leg more.

MENTAL STATUS.—The patient's general attitude was one of general weariness of medical attentions of any kind. She appears to be mentally normal aside from this apathetic state, quite natural under the circumstances.

CRANIAL NERVES. *Optic Apparatus.*—An examination on May 29, 1918, by the Eye Department under homatropin gave R. $20/20 + 7.00 + 2.00$ 180°, L. $20/20 + 8.50$. The fundi were normal. The direct and consensual pupillary reactions to light were normal. The pupillary reactions to convergence were normal. There was no strabismus, no limitation of eye movements and no nystagmus.

Facial.—The facial movements were normal excepting some slight choreiform movements shown in maintaining the cheeks puffed out. The face looks emaciated.

Hypoglossus.—The tongue movements were normal except some choreiform movements.

SYSTEMIC.—There were keloid scars on the left shoulder and right lumbar regions, also a scar on the right abdomen.

The heart showed a slight presystolic roughness.

LABORATORY TESTS.—A report on the urine, May 20, 1918, was as follows: Amount 70 c.c. Turbid. Specific gravity 1.016. Reaction acid. Color straw. Albumin (heat and nitric) negative. Glucose (Fehling) negative. Indican negative. Acetone negative. Large quantity of bacteria.

In this case general medical conditions appear to be much more important than strictly neurological ones. The patient evi-

dently has a chorea and there are evidences of the rheumatic and cardiac conditions which may accompany or precede this disease. Although there appear to be some sensory changes, they are slight, not consistent, and may be explained, in part at least, by the patient's physiological condition, her general indifference or lack of tone, and her slowness of reaction. If there be any real sensory disturbances it would seem that they might be of a hysterical character.

The bladder or other urinary disturbances may be partly neurotic in origin but are so overlaid with real local changes (whether partly due to catheterization, etc., is not clear) that it is difficult, with a rather vague anamnesis, to form a reliable opinion concerning their precise nature. Masturbation may also have played a part in their causation or aggravation. Tuberculosis is another important possibility to be considered as bearing on the general condition.

The patient is an example of that class of invalids who have operations and many medical attentions, but who are apt to drift about, never receiving the complete examination necessary to ascertain their exact condition. Nor are they to blame for this, for there appears to be no institution where ambulatory cases receive such treatment, and there is no coöperation between institutions whereby the results obtained by the various examinations in each can be readily brought together. In the present case a cystoscopy to determine the exact origin of the blood present in the urine has not been made.

DISCUSSION OF DR. STRONG'S CASE

Dr. WALTER A. BASTEDO.—In the discussion of therapy for a case that has been under observation for two years, we must take into consideration the probable fact that the patient has already been treated by the sundry methods usually employed in chorea, and therefore it would be useless for me to go over the treatment of chorea as such. It seems to me, however, from the point of view of an internist, that in the first place chorea requires isolation when it is persistent, and that cannot be accomplished except in a hospital or some home; in a ward it has to be accomplished by screening

the patient. The possibility also of hereditary specific disease should be eliminated or confirmed.

The statement has been made that this patient has been treated with Fowler's solution and with bromides, and that she has received urotropin for the bladder involvement. In the last mentioned condition we are dealing with a localized infection, the urinary tract probably being involved by a pyelitis as well as a cystitis. There may be a relation between this and the chorea; that is, so long as the irritation of the infection continues, she is likely to have recurrences of the symptoms of chorea. Again, the bromides she has received may have a bearing on the paralytic side of the picture, for bromides are believed to have been the cause of paralytic chorea. It is possible that at some time the doses might have been large enough to bring that about, although the recorded amount, twelve grains three times a day, seems not enough. There is a question as to the value of bromide in chorea. In my experience it is not of much service. I have used it in doses ranging from ten grains three times a day to 180 grains a day with only mild suppression of the nervous manifestations, and it has always seemed to me to fail to accomplish very much. Perhaps we are on the wrong track in using bromides. In this condition there is not overtone but undertone, and it seems to me that some other method that would not increase the lowering of muscle tone might be preferable. Aside from bromides, chloral is used a great deal, but I am opposed to its use except in mild doses as a cerebral sedative. One patient, the only case I ever saw with exitus lethalis, was getting 120 grains a day, and my opinion is that the chloral was the cause of the exitus. There was no relief from the choreiform disturbance.

The arsenic treatment is not so generally used today as it was formerly, because many consider that the patients get along just as well without it, and there have been a number of cases of poisoning. In my opinion, this is because generally it is not given in the right way. In using Fowler's solution, it is the custom to increase the dose every day until some poisonous symptoms are manifested. At that stage the patient is poisoned and harm

is done to the organism. Why Fowler's solution should be the only preparation of arsenic used in that generally increasing way, while other preparations are not so increased, I do not know, unless because it was so recommended in the literature a number of years ago. My belief is that this method is wrong, and that we should never go so far as to produce the arsenic symptoms. For intensified arsenifying purposes we have salvarsan; and I believe that if Fowler's solution has a virtue, then salvarsan has a greater virtue. If the latter has not been tried in this case, I should advise it.

If I were taking this girl under treatment now, I would isolate her; give her prolonged warm baths; let her have some little things to amuse her, but no playmates. I would pay great attention to the digestive tract to try to nourish her, as she is obviously undernourished, and that would include particular care to keep the intestinal tract from poisonous products. Then I would give her mild doses of sedatives, chiefly late in the day. If she could be given the cold air cure in an isolation room, it might be of some value. In addition, I would give her a series of treatments by salvarsan, administering a dose of three decigrams once a week for four or five times. At the same time I would have the urinary tract thoroughly cystoscoped and some conclusion arrived at about its exact status, and have the condition treated as intensively as possible, either locally or by vaccines and urotropin, the use of the latter depending on whether her stomach handled it well or not. I would not use bromides in large quantities and would not use chloral at all. I would try magnesium sulphate, by hypodermic at first, in dosage of one cubic centimeter of 25 per cent solution three or four times a day; and, if that did not act well, intravenously. That is what I would do to start with. The question of how much to give intravenously would depend on observations made while using the hypodermic method, the ordinary practice for the intravenous route being to use a six per cent solution until some depression of respiration is noted.

DR. A. L. GOODMAN.—The case presented here exhibits a multiplicity of symptoms combined with choreiform movements. I have

not examined the child, but from the choreiform movements the case does not impress me as one of true chorea, but as being due to some trouble of the pluriglandular system. I note that there is a distinct fullness of the neck in the region of the thyroid. We see these cases every now and then, and usually they are caused by inactivity of the thyroid gland rather than a hypersecretion. Hanneborg, in a Scandinavian medical journal, reported about 69 cases of chorea treated with thyroid gland. Biedl, in his text book, refers to epithelial bodies found in the thyroid of choreic individuals. In this particular case it seems to me that the previous treatment has been unsatisfactory; I believe this is due to the fact that the medication usually employed in chorea has been used, and as I said before I do not believe that a chorea exists, but that we are dealing with a disturbed internal secretion, and that the use of thyroid will prove beneficial.

In reference to the treatment of true chorea, I would say that for the past five years I have given up all medication and have used autoserum exclusively. I know that it has not met with much favor here in New York, but I do know that it has been used extensively with very good results in other states and in Canada. At the last meeting of the Ontario Medical Society at Hamilton, I had the privilege of listening to Professor Allan Brown's report of a series of 44 cases of chorea treated with the autoserum, in which he reported 77 per cent of cures. It was interesting to note that he treated all cases in the out-patient department of the hospital with no untoward results.

During the past five years, through refinement in our technic, we have been able to eliminate many of the unpleasant symptoms encountered in the beginning of the treatment, which were (1) rise in temperature; (2) headache, and (3) vomiting. A rise in temperature after an injection of autoserum is due either to the presence of drugs in the serum or to contamination of red blood cells. No serum should be collected from a patient and used intraspinally, if that patient has been receiving internal medication, until five days have elapsed from the time of discontinuing medication. Blood in the serum will also cause

a rise in temperature. To eliminate red blood cells from the serum, the collected blood should be centrifuged, no matter how long, until the serum is perfectly clear. Another cause of a rise in temperature would be the lack of asepsis. Headache and vomiting are due to the injection of too much serum into the cerebrospinal canal. In using the syringe method, any one, with a little experience, can feel a distinct resistance after a certain amount of serum has been injected, and when this resistance is felt, no more serum should be forced into the cerebrospinal canal. The quantity of serum to be injected varies in each individual case. Headache and vomiting are not serious complications, and usually disappear within a few hours. Very serious symptoms, however, may result from the injection of drugs into the spinal canal of patients receiving medication at the time when the serum is taken. Drugs, injected into the spinal canal, have a very much more potent effect than when administered in any other manner. This is particularly noticeable with salicylates and opiates of any kind.

We do not inactivate the serum, as we did formerly, but immediately after centrifuging it, we place it in the icebox until we are ready to make the injection. In reference to the magnesium sulphate treatment, which originated in Italy, where it was used intraspinally in a 25 per cent solution, 10 c.c. for each kilogramme of body weight, this treatment has been tried and found unsuccessful. I have also tried the simple withdrawal of the spinal fluid in a number of cases without improvement. This was first suggested by Passini in 1914, one year after I began my chorea work. If the theory of the serum treatment stands investigation, it is due to an infection with antibodies circulating in the blood. Certain changes take place in the choroid plexus which do not admit of osmosis of the antibodies from the blood into the cerebrospinal fluid. Results with the serum treatment are far more satisfactory than those obtained by any other form of treatment that has been suggested. Only this last summer, Langley Porter, of San Francisco, used horse serum with very poor results in seven cases of chorea, which he reported before the American Pediatric Society.

The number of cures that I can demonstrate agree with the figures of Dr. Alan Brown of Toronto. By a cure I mean cessation of all choreiform movements within the first week after injection. Another advantage of the autoserum is the fact that when administered early, it prevents, I believe, the heart complications so frequently seen with this disease.

We have made numerous investigations, of both a chemical and biological nature, of the blood and spinal fluid of choreic patients but have never been able to demonstrate any characteristic changes.

PROFESSOR C. C. LIEB.—The depression following the parenteral administration of magnesium sulphate is due to the decrease in the permeability of the cell-wall caused by the magnesium ion. It is known that stimulation of a cell is associated with an increase in the semi-permeability of its cell membrane, and depression with a decreased permeability. Though the aliphatic narcotics (alcohol, ether, chloroform, etc.) and the magnesium ion both produce depression of the cells, they probably act on different constituents of the cell envelope. The oral administration of magnesium sulphate fails to produce depression, because the salt is not readily absorbed from the intestinal canal. While all cells are depressed by magnesium sulphate, those of the central nervous system seem especially susceptible; the activity of nerve fibers and the irritability of sensory and voluntary motor nerve endings are also lessened. It has been suggested that this curare-like action may play an important part in the motor paralysis that follows the intravenous injection of magnesium sulphate.

DR. FREDERICK TILNEY.—So many times we get these cases well, then they go back to their homes and later are either brought to us or come with a recurrence. There is a possibility that something in the home environment, the social environment, is important. Should we not give that matter our attention? Should we consider these cases completely treated if neglected on this side of the question? I do not believe so. We have made arrangements for Mrs. Dwight to look after this aspect of the cases that come here for treatment.

DR. JOHN H. NOLAN.—I have seen Doctor Goodman's work at the Lenox Hill Hospital and he has given me the privilege of using the serum. I have treated a number of cases, and it is marvelous to observe the rapid improvement. One case in particular, sent from the Vanderbilt Clinic with violent manifestations, began to improve within 20 minutes after the administration of the serum and cleared up in a few hours. She relapsed one month later, but was cured with one more treatment. I have never seen any treatment work with such rapidity as the autoserum.

DR. S. P. GOODHART.—I should like to ask Dr. Goodman if he differentiates in these cases between functional or psychic chorea and infectious chorea, and if he treats them all alike? Has he been able to account for some of the features in the cases in which there is no lesion, the cases we speak of as psychic?

DR. GOODMAN.—In reference to Dr. Goodhart's question, it is difficult to make a positive differential diagnosis. We are guided both by

examination of the patient and the previous history. Many of the cases which do not respond to one or more injections of autoserum, are, I believe, due to various so-called habit-spasms, mentally defective types with choreiform movements, and a few so-called psychic cases. Even if a case is not a true chorea, the choreiform movements are often favorably influenced and become less violent, and the patient is made more comfortable by the use of the autoserum.

DR. W. A. BASTEDO.—We tried serum-therapy in two cases and it was a failure. Magnesium sulphate given in the same cases worked much better, but only temporarily.

DR. TILNEY.—We had a similar experience with two cases which Dr. Nolan and I saw at the Presbyterian Hospital. They were chronic cases of distinctly intractable chorea. They were given two injections intraspinally of autogenous serum, but there was little improvement. We put it down to a fault in technic.

CONGENITAL ATHETOSIS

BY LOUIS S. ARONSON, M.D.

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When one reviews the various terms used in the different forms of disturbances of motility, such as tremors, tics, spasms, convulsions, choreic movements, the motor disturbances of Parkinson's disease, of Wilson's disease, of paramyoclonus, and of athetosis, one finds that the conditions are usually clearly stated. Each movement is a distinct class entity, thus deserving its appellation on account of its specificity as to location, time, repetition, severity, quality and duration, as well as its distinct kymographic tracing. But the lines of demarcation between these movements are not always clear, and they frequently merge. At times one type resembles another, at times separate types may coexist in the same patient. Hence there arose the need of different labeling and numerous new adjectives crept into use. Paretic took on the meaning of partial paralysis, or of a tendency towards or similarity to, a paralytic state. And so we find such terms as choreic and choreiform, and what I wish to dwell on mainly in this paper, *athetosis*, and those movements that are usually alluded to as *athetoid*, or as some will have it, *athetotic*.

Hammond in 1872 first described what he then thought was a disease per se and named it *athetosis*, from the Greek *Atheros*, meaning "without fixed position." The chief characteristics of these movements are that they are fixed and of a very definite formula as to character, rate, location, degree. They are timed as to a certain mechanical significance, they are predictable; and as I wish to argue that they are of cortical origin, I will add that they are imitable. It is almost impossible to demonstrate to students what a tic is as you find difficulty with the time element, or choreic movements because they are so widely distributed. The antagonistic action of a synergic group as in paralysis agitans can be shown by a healthy subject more easily, but the typical

athetosis is the simplest of all to imitate by will, and yet the most difficult to find in any explosive states wherein disturbances of muscular action occur, such as epilepsy and its kindred states, eclampsia, infantilis or obstetrical, anesthesia, chemical or hypnotic, or uremia. In these conditions one may find movements that resemble convulsions, spasms, tics, or choreic movements, but one will not find any of the so-called *athetoid* movements. True *athetosis* seems to be a different thing from what is usually called *athetoid* movement. Some cases of *athetosis* show marked muscular hypertrophy. While *chorea* may leave something in its trail, such as rheumatic manifestations in other parts of the body, *athetosis* bears no relation to any other disturbance in the economy. *Chorea* may be checked by saturating the system with arsenic; *athetosis* has never been arrested, except temporarily by Hammond and Morton, who stretched the median nerve, and by Erb, who gave large doses of hyoscin. There is another point of dissimilarity to *chorea*, and that is the movements never cease even during sleep. Some authorities, however, claim to have seen *athetosis* cease during sleep (Strümpell). I have thus tried to enlarge von Monakow's differentiation between *hemichorea* and *athetosis*, which was, in the main, an anatomical one. That *chorea* occurs so often in the face is to me of no special value as a point of differential diagnosis. The case at hand might be one of choreiform movements limited to the distal parts of the fingers, and the question of similarity to *athetosis* might be raised. From the other disturbances of motility enumerated, the diagnosis is at all times simple, except possibly the tremor of paralysis agitans type. Personally, I believe that *athetosis* is a compound agitans movement. By that I mean that each finger moves independently, like a Parkinson movement in that it is flexion-

extension or synergic disturbance. Paralysis agitans has so many other features that the tremor is not paramount in making the diagnosis; it may even exist *sine agitatione*.

This case is presented to this conference for two reasons; first, on account of its relative rarity clinically; second, because I wish to state some views as to its pathogenesis, despite the fact that I expect these views to be disputed.

This boy is 20 years of age, a laborer, native of the United States, of Irish extraction. He has no subjective complaints. As a matter of fact, I saw him at the local board rooms where I happen to be the examining physician and invited him to come here. He has a speech defect, a sort of dysarthria, which possibly is concomitant with his athetosis, as he has never learned to use 50 per cent of the sounds necessary in speech. It is remarkable how his friends can understand him, because at first his speech appears a slurring or jargon. C F G H J K P S V X and Z are mispronounced, and the errors are always absolutely alike. He pronounces C as D, F as epp, G as D, H as ate, J as day, P as B, Q as jew, S as ets, T as D, V as B, X as eggs, and Z as D. In other words he has not learned the use of his linguals and labials. His speech defect, like his motor disturbance, is due to a lack of cortical innervation to his periphery. The patient presents constant motion in all the five fingers of his right hand. These movements are uninterrupted, rather slow, rhythmical, monotonous and resemble the action *in toto* of what occurs in an individual muscle when the sinusoidal electric current is passing through it. His fingers seem to be aimed at a definite purpose, such as seizing and manipulating a screwdriver, but their movements appear comical or extraordinary, in that they overdo what they set out to do. When they flex, they overflex, and when they extend, they overextend, so that the articulations are rather loose. They follow the same order, so that, beginning where you wish, they are the first finger, then the fifth, and finally the second, third and fourth, in succession.

His family history throws no light upon his condition. His father died of apoplexy and his mother of tuberculosis when he was a small

child. They had seven children. Four are alive and well, three died. There were no miscarriages. I could not ascertain the causes of death of the three that died, but two were twins, and had a bad start. The boy, until two years of age, was taken care of by an uncle. After the death of the latter the child was taken to a Roman Catholic orphan asylum, where he remained until able to go out to work. There were apparently no cases of epilepsy, insanity or alcoholism in his direct ascendants or his collaterals. His development was normal though a little retarded. He was never sick. At one time he had a slight discharge from his right ear, and it has slightly diminished his acuity of hearing in that ear. His habits are good. He uses very little tea, less coffee, hardly any tobacco and no alcoholic beverages. He sleeps and eats well, weighs at present 132 pounds, which is his best weight, and is an active, strong, healthy boy. Out of necessity he grew up left-handed, but he dresses himself easily and does other acts that show he has no dyspraxia in his left hand. But if I ask him to pick up this pin, you see that there is considerable difficulty, because he cannot stop his athetosis. As a matter of fact I am sure that the application of his will increases it.

There are no abnormal attitudes or deformities save for his athetosis, and the trick that he employs to conceal it. He has learned that he has a slight power of flexion in his thumb, or rather that the extension is a trifle weaker than flexion, and he can so manipulate his fingers by the use of his left, or well, hand, that he can overlap them as shingles and hold them down with his thumb. This is the only time that his constantly working fingers get any rest. He holds his head slightly rotated towards the left. His face has a peculiarity worthy of mention. Like the celebrated masked condition of Parkinson's disease, athetosis seems to be accompanied by a distinctive facies. His face is taut but not masked. There is a little play in the upper part, so that when he laughs, he laughs only with his eyes. The muscles supplied by the facial nerve seem to be rather rigid, especially on the right side, but the rigidity seems more marked in the muscles supplied by the lower fibers than in the muscles supplied by the upper fibers. He

puckers his cheek as if he were trying to dislodge a small piece of meat fiber from between his teeth, by manipulation and buccal suction. He shows no disturbances of gait or equilibrium coordination. The non-equilibrium coordination tests he does very well, considering that he can use his right hand but little, as the athetosis prevents him from using the entire extremity in any other capacity than that of a shovel or spade. There is adiadochokinesia in his right hand, and diadochokinesia in his left. This I feel quite sure is not of cerebellar origin, but due to the fact that physiologically his right hand is the same as on the day of his birth. By this I mean that he has not educated it to that inhibitory control to which he has trained his other hand. His writing with his left hand is fairly good. There are no changes in any of his reflexes, they are equally active, as they should be, and the same is true of both superficial and deep reflexes. Outside of his athetosis there are no other disturbances of the voluntary motor system.

The rate of his athetosis is, on the average, 44 per minute. The regularity is deserving of note. Whether fatigued after work or fresh from sleep, the finger movements are apparently always of the same rate and rhythm. There are no disturbances of muscle strength. Each individual finger is as strong as that of the opposite side, when once it is harnessed. But it is hard to harness the finger because of its flexion and extension. Considering the fact that he is left-handed, we expect his right hand to be weaker than his left, but there is no indication of any post-paralytic weakness. On the Duchenne spring manometer he had 80 pounds in his left, 75 in his right, and at another observation 60 in his left and 55 in his right. This I think is to be considered normal. Hirt's assertion that there are skin temperature changes, and that on the normal side the temperature may be .5 to 1 degree C. higher than on the affected side, could not be verified in this case, although I looked for it several times with a specially constructed spiral surface thermometer.

On the abnormal associated movements that occur, post-hemiplegia and other allied phenomena, I spent considerable time. I found the

radialis sign of Strümpell present on his right side and absent on his left. This was the only one positive. All the rest were absent on both sides. In the upper extremity there was no evidence of the presence of the pronation signs of Strümpell, Babinski or Bechterew. The thumb sign of Klippel-Weil, and the interosseal phenomena of Souques could not be tested. In the lower extremity he had none of the signs of Neri, Babinski, Grasset-Bychowski, Raimiste, Strümpell, or Marie-Foix. In the lower extremity on the right side he exhibits a very mild, feeble athetosis similar to that of his hand.

There are no sensory disturbances anywhere, either epicritic, protopathic or of deep sensibility. His cranial nerves are all entirely normal. His fundi are normal, so is his acuity of vision, which is 20/20 in each eye. One pupil can be said to be slightly larger than the other, and also slightly irregular, but one must have very good eyesight himself to see it. His cranial morphology is normal. There are no pigmental, vasomotor or secretory disturbances, no arterial or venous stases, and no glandular disturbances that one can see or feel.

His blood pressure is 130 systolic, 85 diastolic, Tyco's instrument, auscultatory method. His radial pulses are equal, soft and regular, 78 per minute. His heart sounds are slightly distant, though there is no arrhythmia and no murmurs. The apex beat is where it should be. His gastro-intestinal, respiratory and genito-urinary systems are all normal. The skeletal system is entirely normal except for the right upper extremity. Here the shoulder and elbow joints are negative, but the wrist joint is a little hypertonic, and there is a tendency towards extension. In most cases there is a tendency towards flexion.

Though the disturbance is in the fingers, which seem to be in continuous flexion and extension, there appears to be at no time any lateral movement or spasm, so that I think it is safe to say that the muscles of abduction and adduction, the interosseal, are not involved. Hence we have a simple paralysis agitans type tremor, or antagonistic contraction, except that it follows in all the other fingers at a given rate.

The results of autopsy findings vary, hence

the theories of origin vary. Charcot taught that besides the posterior nerve fibers in the corona radiata there is a bundle of fibers that possesses peculiar motor properties. Gowers regarded it as an affection of the optic thalamus; Anton, of the lenticular nucleus and corpus striatum; Eulenburg, of the cortex; Eisenlohr, of the spinal cord, and Pineles, of the cerebellum. Kahler and Pick claimed that all attempts at localization were futile, as the disturbance might be at any area of the motor tract. Déjerine and Solhier, after a careful autopsy, found nothing at all. Gordon Holmes (*Brain*, 1904) advanced the theory that a lesion exists in the region of the basilar nuclei, particularly of the optic thalamus or the subthalamic region, the most probable explanation being that it disturbs afferent pathways, particularly those that run between the cerebellum and the red nucleus, and that lesions of the motor cortex and those which involve the pyramidal tracts have little or nothing to do with it.

In a recent number of the *NEUROLOGICAL BULLETIN*, J. Ramsay Hunt set forth a new theory. If we accept this theory, and for the present we cannot but accept it, then we assume that the movement disturbances of paralysis agitans are due to a break in the pallidal system fibers of the corpus striatum, thus releasing automatic control in the hypothalamic region, the extrapyramidal tracts of the spinal cord, the rubrospinal and other descending tracts. Man is thus conceived as possessing two distinct mechanisms for the control of motility, first, the pyramidal or corticospinal, which is for the control of isolated and synergic movements, and, secondly, the striospinal, which governs automatic and associated movements. In athetosis we find no disturbances of the associated movement type. Just as Hunt regards paralysis agitans as a syndrome that occurs at this given anatomical landmark, whether the lesion be inflammatory, hemorrhagic, atrophic, gummatous or neoplastic, so with athetosis. It may follow a variety of lesions, disturbances between the thalamus and the cortex. This is of course hypothetical, but so are all the other theories of pathogenesis that I have enumerated above, and some of them were based, not

on psychological or physiological logic, but on actual postmortem facts.

In sleep, whether normal or induced by anesthesia or hypnosis, a number of irregular muscular twitchings and contractions independent of those necessary for the work of the respiratory and circulatory systems, can be seen. In hysteria, in catatonia and in fakir's trance there may be for a while an apparent absence of all skeletal movement, but close observation may show that the muscles are constantly undergoing fibrillary and sometimes larger contractions. In the healthy, waking state the musculature is never for a moment at rest, but the fallacious dictum, *mens sana in corpore sano*, has a little truth, in that education seems to bring about a more rapid, more complete, muscular control. Watch the little girl just learning to write. The attempt at control of the muscles of the hand and forearm is with her a painful process. Her spine straightens out, she bends her head to the opposite side and possibly draws down the angle of the mouth on the same side. After a little practice all these associated movements are no longer required, the cortical pathways have been established, and are better fitted toward their newly acquired function. Good inhibition of purposeless movements has come into existence.

The infant at birth is a mass of movement from the moment that it utters its initial cry. There are groups of muscles which with us are still constantly overworked, for example the various intrinsic and extrinsic muscles of the eye. In the infant which fails to develop an education of its cortex, possibly from an agenesis corticalis, we see the idiot with all sorts of movements, so well known, as head shaking, and constant spinal flexion and extension. Most of the cases of double athetosis come under this heading. The profession is fairly convinced that double athetosis is a cortical disturbance, but is rather slow in accepting the view that unilateral athetosis is also a cortical disturbance. The man of maturity and education can get along with less sleep than his simpler brother. Morons sleep like the dead, showing scarcely any movements during sleep. The comical gesticulations that foreigners, especially Latins and Semites, show in our clinics might at first be conceived as due to a failure

to comprehend our language. But as these races yield a goodly percentage of our neurotics, some of their gesticulations may be due to an improper cortical control of muscle inhibition. I am told that in the army one can readily see the ease with which our studious youth falls in for discipline. Yet the late Gen. Bell had great difficulty in his task of forming the Argonne regiment from the 77th, made up of boys selected from the lower East Side, where there is a decided neurotic trend. I might point to this as an example of what could be done with the neurotic by training his cortical control. I think it is safe to say that we are born with a tendency to all sorts of irregular and involuntary movements, and that as we proceed, from our first sensations of receiving light, to fix the stare or gaze, we begin to learn to control our cortical inhibition. Some are inclined to the view that there must exist a center of inhibition. Whether or not this is so, I cannot say. But I feel that man must have developed in his cortex, and from there to his midbrain, a system of fibers that came late in evolution and that appear late in infantile development. This system spells inhibition for all purposeless, irregular, unmeaning and uncontrollable movements that man, by education of his central nervous system, including his cortex, was obliged to evolve for knowledge and for manual dexterity.

It is to these regions, therefore, that one should look for a lesion in this case, if anatomical opportunity presents itself,—something in the way of a failure of cortical development, an agenesis cerebri, possibly racial, familial, Mendelian; possibly and more probably some prenatal inflammatory process, as a minute hemorrhage, with what goes along with it, absorption, and cystic formation, may exist.

Such cortical bleeding may be due to the tuberculous diathesis that existed in the mother, or the anemia and death by earth apoplexy of the father. The question of syphilis has not yet been answered to my entire satisfaction, nor have the kymographic tracings been made as yet. They will be made as soon as time and opportunity permit.

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CLINICAL SIGNS IN NEUROSYPHILIS

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I have learned by experience to lay little weight on the statement of patients regarding the history of early syphilis. It is surprising how many state that they never had a primary lesion, but when I ask if they have had gonorrhœa they reply to me affirmatively. Of course it is well to bear in mind the possibility of intra-urethral chancres; their independence of gonorrhœa or association with it. Often, if we interrogate patients who come with late manifestations of syphilis and ask them if they had a secondary rash on the skin; they may reply that they never had an eruption on the skin or involvement of the mucous membranes.

Early skin manifestations of syphilis are sometimes very transient. The initial lesion also may be insignificant or occur on extra-genital parts of the body. Chancres of the lip, the tonsils, the fingers or other parts of the body are of rather frequent observation. In medical men, infections of the finger during their professional work are not rare and are often wrongly diagnosed.

Enlargements of lymph nodes occur chiefly in the early period of the disease, and under modern therapy they usually disappear within the first three to six months. Enlargements of the epitrochlear lymph nodes are met with in individuals with occupational dermatitis of the hand and forearms, and under such conditions are of no significance in the diagnosis of syphilis.

In all our cases of early syphilis we pay special attention to the condition of the pupils and deep reflexes so that we may have data for comparison later in the disease. In the secondary period we are more apt to find slight pupillary irregularity with sluggishness rather than the typical Argyll-Robertson pupils. We encounter, however, involvement of the central nervous system in early syphilis at times without pupillary changes. In a later period of syphilis, changes of the pupils are a most important diagnostic aid, and are some-

times the only indication of involvement of the central nervous system.

In paresis we are more apt to note irregularity in the size of the pupils associated with exaggeration of the deep reflexes. In tabes, of course, we encounter the more typical Argyll-Robertson pupils. In chronic alcoholism I have seen fixation of the pupils and have been able to eliminate syphilis by serological examination of the blood and spinal fluid. In such cases it is, of course, well to bear in mind the possibility of abortive forms of tabes in which the blood and fluid are also negative.

I am at present very much interested in the following family group of cases.

About one year ago a physician in Springfield referred to me a woman with Argyll-Robertson pupils. She was a teacher in a college and consulted an oculist in Springfield who referred her to me for further examination. The blood and spinal fluid were negative. She brought to me a sister with a typical ataxic gait who also showed the same pupillary changes. She stated that she had a brother with an advanced ataxia and another sister in the same condition. Her father had died in an asylum from some form of mental trouble. In two of these cases I was able to eliminate syphilis by an examination of the blood and spinal fluid and concluded that all of the patients had Friedreich's ataxia.

In tabetics who present objective manifestations of the trouble we find as a rule in our serological work that about 30 per cent have negative Wassermann reactions in the blood and spinal fluid. In the parietic type fully 99 per cent have both positive blood and fluid. A positive Wassermann in the blood with a positive Wassermann in the high dilutions of the fluid, together with a strong globulin reaction and a Lange parietic curve, point to paresis even in the absence of the stigmata of degeneration.

I would like to emphasize here the significance of a positive Wassermann reaction in the so-called latent period of the disease.

Many practitioners treat cases for one, two, or perhaps three years and at the end of that time if the Wassermann reaction is negative, well and good; if positive they consider the patient potentially cured.

The more we know of syphilis the more we must believe that a positive Wassermann reaction means the presence of active spirochætae in some part of the body.

Warthin is entitled to a great deal of credit for his careful pathological work in visceral syphilis. He has found in the heart muscles, the pancreas, the kidney, the spleen and the testicle, foci about the small blood vessels which are identical with those we see in the primary lesion and the secondary skin manifestations. These lesions may exist for years without producing much if any functional disturbance of the involved organs, but are sufficient to produce a positive Wassermann reaction, and if allowed to progress lead to marked degenerative changes. Furthermore, a persistent positive Wassermann reaction, in spite of prolonged and intensive treatment, at times is found in patients who subsequently develop neurosyphilis. In these patients it is often possible to obtain a negative reaction in the blood, but when treatment is suspended the reaction again becomes positive. In such patients the blood is reinfected from the disease in

the central nervous system and cannot be controlled without intraspinal medication.

It is, therefore, important to keep in mind the significance of a positive Wassermann reaction, as the future welfare of the patient depends on obtaining a persistent negative reaction in both the blood and spinal fluid before we can pronounce a cure of the infection.

In the diagnosis and prognosis of syphilis we rely more on our serological tests than on the physical signs which the patient presents. It is important to correlate our clinical observations with our laboratory findings. A trained observer is able at times, by noting the appearance of scars of pigment changes of the skin, to say whether or not such a patient has had an old syphilis, but there are so many other conditions which produce scarring and changes of the skin that one is apt to be misled by a dependence on objective signs alone.

In congenital syphilis a diagnosis can frequently be made by the characteristic scarring about the mouth, the appearance of the nose, the teeth, the formation of the skull and changes in the long bones, but in acquired syphilis not infrequently all the external manifestations disappear. In such cases we should be guided as to the cure of the patient or the presence of the disease by our properly controlled serological tests.

CLINICAL SIGNS VERSUS COLLOIDAL GOLD

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The patient, J.B. (23065), is a man 33 years of age, married, by occupation a fireman. Date of examination, November 13, 1918.

HISTORY. Present Illness.—The patient complains that he feels dizzy all the time, except when lying down, and that he has poor control of his legs when walking. These symptoms he has noticed for the past two weeks (preceding November 13). Besides this he has also noticed some slight trouble with his arms, e.g., when picking up a glass of water he sometimes spills it.

Since the above symptoms appeared he has noticed also that if he looks a long way in a car he doesn't get the signs as well as he used to.

The dizziness and poor control began about two weeks before November 13, coming on for about two or three days and the visual disturbance since then. An improvement in all the above symptoms soon began, however, and continued up to about December 1, when they had, according to the patient, disappeared.

He at first said he had had no pains nor headaches except for 10 or 12 days before his trouble began, when he had pains in the head every day. These stopped when the trouble (dizziness, etc.) began. There has been no vomiting, but some slight nausea in the morning since the trouble began.

On subsequent questioning, however, the patient recalled that he had had for a number of months occasional sharp, shooting pains in the legs lasting a second or two. These were not very noticeable. He particularly remembered pains in the left instep after the paralytic stroke mentioned below. These were better at the time of examination.

The patient also complains of a sore throat, a hoarse voice and a sore in the nose. The sore throat he has had since February, 1917. It is caused by "cankers." A doctor had evidently told him they were specific. A recent examination by Dr. Osgood, however, shows only a

chronic follicular tonsillitis, non-specific in character.

The hoarse voice he has had for four months. (See also Vagus Nerve.)

The sore in the nose the patient dates from an operation for adenoids some three months previous. This latter, however, is not apparently connected with the nasal lesion found by Dr. Osgood (see Nose).

PREVIOUS HISTORY. Previous and Present Habits and General Physiological Status.—The patient has not used alcohol for the past ten years, previous to that an occasional excess. He sleeps well; his appetite is good; no fever has been noticed and he has had no disturbance of urinary or sexual functions.

Diseases, Injuries, etc.—He has had no injuries. He had, as mentioned, an operation on the nose for adenoids three months earlier.

He admits a luetic infection 11 years ago followed by incomplete treatment.

On May 19, 1917, at a fire, he was working with a company in a cellar full of illuminating gas. The patient felt what he thought was a cool breeze and snuffed it up. He was afterward told that illuminating gas felt cool. He noticed no odor but the place was full of smoke. He felt faint for a few minutes when he came out into the air and took hold of a fence to keep up. This was at 3 A.M. He then went back into the cellar, helped to wash down and felt all right. He went back to quarters, made an entry in a book and immediately afterwards noticed he "only had one arm," i.e., he could only partially raise his left arm, couldn't use his left fingers, couldn't even open a cigarette box. This was about 3:20 A.M. The arm began to feel better at once. In about three weeks he had full control of it (i.e., could make nearly all movements) but not full strength. For months afterward he noticed a weakness in the left arm and hand and even now they are more easily fatigued and feel weaker than formerly. This is more noticeable when he is lying down.

Another man working in the same cellar had. to take the rest of the day off on account of feeling sick and having a headache, but he had no unpleasant after effects. The patient had been exposed before to gas when others had been knocked out but had not been affected himself.

Family History.—He has been married nine years and has two living children, one eight and one three years old, both healthy. No deaths, stillbirths or miscarriages. His wife is healthy.

VOLUNTARY MOTOR SYSTEM. *Gait.*—A slight occasional uncertainty in gait was noticed, especially when walking on a line.

Coördination.—The patient is right-handed. There is a slight swaying when standing with the feet together and the eyes open which was somewhat increased with the eyes closed. There was thus a rather doubtful Romberg.

The finger to nose test was quite well performed with the eyes open but there was some uncertainty with the eyes closed, especially when done quickly with the left hand. The heel to knee test was done fairly well but when done rapidly there seemed to be some tendency to overreach.

There was perhaps a slight dysmetria shown in the pronation-supination test.

All the above disturbances appear to have diminished or disappeared since the earlier examinations.

Skilled Acts.—His handwriting was normal, but the patient said he had noticed some unsteadiness when his trouble was worse some time before.

His speech was normal.

Abnormal Involuntary Movements.—No abnormal involuntary movements were noted.

Reflexes.—(o=absent, 1=slight, 2=active, 3=very active.) (a) *Deep.* Pectoral: R. 2+ < or =? L. 2+; biceps; R. 2 < L. 3; triceps: R. 2 or 3 < L. 3; radial R. 3 < L. 3; ulnar: R. 2 < L. 2+; suprapatellar R. 1 < L. 2; patellar R. 1? (on reinforcement 1+) < L. 2; Achilles R. (on reinforcement) 1 < L. 1+ or 2, periosteal (shin) uncrossed R. o, L. present; crossed patello-adductor ("crossed adductor") present R. to L., not present L. to R.

There are thus two phenomena here: an

increase on the left as compared with the right and a diminution in the lower extremities as compared with the upper on both sides.

All the deep reflexes were hypotonic, even those which were indicated as very active. There is no clonus.

(b) *Superficial.*—The ciliospinal appeared to be present on the right, more doubtfully so on the left. The abdominal reflexes were present but were not well marked. The cremasteric was more marked on the right than on the left. There was no Babinski nor any of its modifications.

Muscle Strength.—This was normal in all parts with a slight doubtful exception of flexion of left leg. The right arm was stronger than the left, but hardly more than would be due to right-handedness.

MUSCLE STATUS.—No change in tonus was noticed except as noted above in the character of the reflexes.

ABNORMAL ASSOCIATED MOVEMENTS.

GENERAL SENSORY. *Touch.*—This was normal (only acuity was tested).

Pain.—There was a doubtful diminution, especially, perhaps, near the Achilles tendon and on the hands.

Vibratory.—This was normal on the big toe, ankles and shins, but there appeared to be some diminution on the elbows.

Muscle-tendon.—Was normal in all parts.

CRANIAL NERVES. *Olfactory and Nose.*—From the nasal septum Dr. Osgood removed a broken-down gumma. There was a slough to the bone. Another examination on January 3 showed a great improvement, in fact it was practically healed.

Optic Apparatus.—The fields were normal.

The fundi were normal.

The left pupil was somewhat smaller than the right, and the left seemed somewhat irregular. The right pupil reacted normally to light, the left showed a diminished reaction to light. Both reacted to convergence, but possibly the left less than the right, so that a left Argyll-Robertson pupil was somewhat doubtful. The movements of the eyeball were normal. An examination by Dr. Holden, December 23, 1918, was as follows: R. pupil, 3.5 mm., reaction

fair, L. pupil, 3 mm., reaction sluggish. Irregular. Acuteness of vision, fields, fundi and mobility normal.

Acoustic Nerve and Ear.

Trigeminus and Mouth.

Facial Nerve and Face.—The facial movements were normal. There was no tremor.

Glossopharyngus and Vagus Nerves, Pharynx and Larynx.—The voice was hoarse and Dr. Osgood found that there was a left vocal cord paralysis. Another examination on Dec. 23 showed no change from this condition.

There seemed to be a tremor of the palate when the mouth was open.

The Hypoglossus Nerve and Tongue.—The position of the tongue when protruded was normal. There was no tremor. The tongue movements were normal.

Mental Status.—This appeared normal as to intelligence, emotion, attention and memory.

LABORATORY TESTS: The blood showed a four plus Wassermann.

The spinal fluid showed 73 cells, four plus globulin, four plus Wassermann to 0.2 c.c. and colloidal gold 5555431000.

A subsequent test, made December 2, showed a four plus Wassermann to 0.2 still present in spinal fluid.

The urine was negative.

Treatments.—Beginning with November 20 and ending January 7 he has received six mercury injections (intramuscular), six salvarsan (intravenous) and two intraspinal salvarsans (December 3 and 26).

That we are dealing here with some form of neurosyphilis is obvious, and the principal question appears to be whether we are dealing with a cerebrospinal lues, a tabes complicated with cerebrospinal lues, or a taboparesis. The last named possibility will be considered first.

Perhaps the most interesting feature of the case is the condition of the deep reflexes. This has been found to consist of an inequality, the left side being greater than the right, and a diminution of those of the lower extremities on both sides together with a hypotonia of all. These findings seem to corroborate the anamnesis of a left paralysis of the arm some twenty months ago and to show that we are probably dealing with a unilateral upper motor

neuron lesion and a concomitant bilateral posterior root or posterior column lesion. Which of these overlies the other cannot be definitely ascertained. Clinically the signs of the pyramidal lesion preceded the other, but it must be remembered that the diminution of the deep reflexes may have preceded the other clinical manifestations of a posterior column lesion.

It is well known that in general paresis there are often found cord changes. These consist of either a bilateral posterior column degeneration or a bilateral pyramidal degeneration or both. The posterior column degeneration may be either of the two tabetic types, i.e., a simple secondary degeneration or a systemic degeneration following Flechsig's myelogenetic root zones. The coexistence of the pyramidal and posterior column lesions produces a varying mixture of symptoms, especially hypotonic increased reflexes, regarded by some as characteristic of a certain number of cases of paresis.

We are not, however, dealing with this type of pyramidal lesion here, but with a unilateral lesion of probable sudden onset. As is well known, this latter may also be a feature of early paresis (as well as of later) characterized by a usually rather rapid recession of symptoms and with either an epileptiform or apopleptiform onset. The stroke in the present case appears to have been of the latter character.

On the other hand, we have here quite well defined tabetic symptoms (diminution of the lower deep reflexes, slight ataxia and possibly lightning pains) but no other cortical symptoms than possibly the pyramidal lesion. The absence of any noticeable mental defect or change does not, however, negative a taboparesis, inasmuch as an initial paralytic attack may precede noticeable mental signs by a considerable interval of time, the tendency, however, of such attacks being usually to be followed by a further progressive mental change.

The conditions seen in the deep reflexes, then, do not seem, at first sight, to contraindicate a taboparesis but even rather to favor it. The absence of bladder symptoms and the absence or slight character of lightning pains also favor a taboparesis as opposed to a tabes.

A further consideration, however, of the

clinical signs modifies the above impression. In the first place, while paralytic strokes may occur in the course of a tabes they are commoner after the tabetic symptoms are more pronounced than in this case. The same applies, vice versa, to tabetic symptoms occurring in the course of a general paresis. When we consider the stroke itself more closely, we find that while there was quite a rapid partial recovery there has been a residuum of the attack for a long time. This is not characteristic of the early prearetic seizures. Furthermore—a period of twenty months has elapsed without other parietic signs (mental symptoms or facial tremor) becoming manifest. This, again, is possible but not characteristic. Another possibility which has not been considered yet is whether the stroke may not have been caused by gas poisoning. The patient did not, apparently, experience the more usual symptoms of cerebral congestion, headache, subjective sensory signs, etc., but this form of poisoning varies in its manifestations, a coworker was undoubtedly affected and the causal nexus seems so clear that it seems we must assume that the inhalation of gas was, in part at least, responsible for the attack. This raises the further interesting question as to how far true luetic changes may have made the patient more vulnerable—a question we cannot discuss here.

The vocal cord paralysis is another symptom requiring consideration. How long this has been present is not certain. Judging from the hoarseness it has been present only four months and consequently cannot be referred to the gas poisoning. Vocal cord hemi-paralyses may occur in the course of a tabes, but usually in connection with laryngeal crises of which there is no history in the present case. This symptom appears then to be against a tabes or taboparesis.

The pupillary changes do not appear to be decisive. They are consistent with an early tabes or taboparesis but are perhaps rather more characteristic of a cerebrospinal lues.

Many of the above considerations apply to the second possibility raised, i.e., a combined tabes and cerebrospinal lues. The case for a tabes seems considerably stronger than for

a paresis. Such symptoms as an undoubted diminution of the lower deep reflexes, a probable slight Romberg and other ataxic symptoms with rather slight lightning pains would point to tabes. On the other hand, as pointed out, the absence of bladder disturbances, of any kind of crises and the nature of the vocal cord paralysis are against it. Besides this we note the rather fluctuating character of the symptoms. The undoubted improvement of the patient since November 5—an improvement, by the way, which appears to have begun before recent treatment was initiated—is also rather against tabes. The recent symptoms he has complained of appear to have been of the nature of an attack appearing rather suddenly and then receding.

As far as a cerebrospinal lues, complicated only with a stroke partly or entirely due to gas poisoning, is concerned, the general ensemble of clinical symptoms favors it. The generally irregular character of the symptoms and their apparent amenability to treatment together with the evidence of the “coarser” luetic manifestations shown by the nasal lesion, give the clinical picture more of this appearance.

On the other hand the laboratory findings, especially in the spinal fluid with its colloidal gold curve, point distinctly to paresis. Which ever side future developments favor, whether clinical signs or laboratory findings—especially colloidal gold—the outcome will be equally instructive.

Inasmuch as we are dealing, in tabes and general paralysis, with the same pathological process—and even in cerebrospinal lues with one not very dissimilar—manifesting itself in different parts of the nervous system, naturally many varieties of mingling and intermediate types might be expected. Nevertheless the question whether we are dealing with the cerebrospinal luetic group or with the taboparetic group, and the question whether, if the latter, the case falls under the category of tabes or paresis, are both of great importance from a prognostic and practical point of view. The better prognosis in the case of a cerebrospinal lues of course requires no mention, but even in the taboparetic group whether we are dealing with a tabes or paresis is of great importance. A tabes may not, in

fact usually does not, terminate in a general paresis and may even take a relatively benign course. This apparent self-limitation of the process is of course seen even more strikingly in amblyopic tabes and also in other diseases.

Another feature of the case worthy of mention is the presence of an apparently healthy wife and two healthy children, marriage having taken place about two years subsequent to the infection. It is hoped that a test of their blood can also be made.

DISCUSSION OF DR. STRONG'S CASE

DR. NORMAN SHARPE.—While the colloidal gold paretic reaction undoubtedly occurs in other conditions, such as brain abscess, and it has therefore been concluded by some that this is a test depending on nerve cell degeneration, yet all the cases that I have seen with a paretic curve and with no, or only slight, clinical signs, either were cases of paresis or developed it months afterward. One patient, in my experience, apparently non-paretic, developed paresis clinically four months after a colloidal gold paretic curve, and another developed it in five months. I know some laboratory workers who say the colloidal gold paretic

curve can precede paretic symptoms by ten years, but I hold this is unproved.

I would regard this case rather as one of cerebrospinal lues at present, though the colloidal gold paretic curve would make me watch the patient for months, and I would have the test repeated at intervals by different laboratories. The case certainly strikes me as one of cerebrospinal lues. There is no history of lightning pains, bladder trouble, hypesthesia, lowered or absent reflexes, so one cannot make a diagnosis of tabes. I think that we must have more than one symptom or sign to make a diagnosis, and where cerebrospinal lues will cover it, as in this case, I regard that as the most probable diagnosis.

DR. CHARLES A. MCKENDREE.—This case seems to me to represent one of cerebrospinal lues. I have seen the colloidal gold curve in cases well known to be neurosyphilitic in which there were no signs of paresis whatever. I believe the paretic curve appears also in multiple sclerosis, and therefore I would not pay too much attention to the colloidal gold representation as long as we have no other signs to go with it.

APHASIA AND ASSOCIATED SPEECH PROBLEMS

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(CONTINUED FROM THE NOVEMBER-DECEMBER BULLETIN)

CHAPTER III

Marie²² located the areas for speech, together with those for apraxia, and considered that together these areas were centers for the ideas of highly skilled and conventional acts. According to him, lesions in areas causing apraxia result in a defect of intelligence, as a consequence of which the apraxic patient is unable to perform purposeful acts in the absence of any paralysis, and he also considers that the apraxic patient suffers from aphasia of low grade.

That the destinies of speech and apraxia are intimately bound seems to be undoubted, despite the fact that cases of apraxia have been reported which seem to be limited to the left side only, pointing to lesions in the right hemisphere. The following quotation from Wilson's article, "A Contribution to the Study of Apraxia with a Review of the Literature,"³⁵ will illustrate this particular point, i.e., how closely bound aphasic and apraxic manifestations may be found in the same patient:

"There is a case* of left hemiplegia in the National Hospital at present which presents certain features of unusual interest for our subject. It is a case of mitral stenosis with an ordinary embolic lesion of considerable extent in the right cerebral hemisphere. The clinical picture is one of complete flaccid paralysis of the left arm and leg; the left face is also affected, but to a much less degree. On examining the patient one is at once struck with her immobility and lack of spontaneity. She rarely speaks unless spoken to; she still more rarely makes any voluntary movement

of her non-paralyzed right limbs. When requested, however, she can perform any movement with her right arm in an eupractic way, and there is no agraphia. Her face is more or less 'fixed' in an expression suggesting anxiety; her eyebrows are symmetrically elevated, and her eyes look straight in front of her. This expression never varies. She is quite incapable of making any grimace, of smiling, of laughing; when asked to try, her reply is 'I can't.' She is incapable of closing her eyes on request, or, rather, of sustaining the contraction, for she will close them momentarily, opening them again without the slightest pause. She is unable to look voluntarily to the right or the left, and it is only with difficulty that she can be tempted to gaze in either of these directions, by the stimulus of a sound or otherwise. Yet there is no paralysis of conjugate deviation at all. She can protrude her tongue, can show her teeth, bite, etc., well enough. Her voice, since the stroke, has been peculiarly monotonous and rather shrill; she is incapable of modulating it. There is a fixity of tone more or less in keeping with the fixity of her facial expression, but I have the authority of Sir Felix Semon for the statement that the larynx is normal. She is incapable of making the movements of swallowing, though with food in her mouth she swallows well, if slowly; she cannot cough, or sigh, or yawn voluntarily."

This case of Wilson's, then, illustrates quite definitely that apraxia may occur with lesions in the right cerebral hemisphere. That this cannot in any way be used to separate the problems of aphasia and apraxia is seen from the fact

* A. P., under Dr. Risien Russell.

that many cases of aphasia have occurred with lesions also of the right hemisphere, and that, too, in right-handed individuals of right-handed stock. Two personal cases will be mentioned later.

A systematic presentation of the subject of apraxia as it relates to aphasia is extremely difficult, because of the many contradictions, disputes and uncertainties of psychological, anatomical and pathological knowledge adduced in the elaboration of both subjects. The attempt made by so many sharply to delineate and separate apraxia and aphasia as definite parts of general intelligence is perhaps responsible. Here no such attempt will be made, and the idea followed will be to correlate aphasia, apraxia and general human intelligence closely without particular regard to specific cerebral localization of these functions.

Wilson states that he has seen ideational apraxia and agnosia in alcoholism, senile dementia, delirium, postepileptic confusion, etc. Here "under the influence of some toxin or some general pathological process, the function of the whole cortex must be seriously at fault." We shall also see that Liepmann's¹¹ original case also presented profound mental changes such as are seen in advanced senile dementias. Hughlings-Jackson¹² always insisted that speech "is a part of thought and that in conditions of aphasia thought processes of all kinds were involved." Von Monakow,³⁴ in illustrating his theory of diachisis, assigns a different interpretation to those defects of speech which are seen after a lesion and which he says are slight in degree and soon pass off, and those which remain well marked and permanent. The early symptoms, which are transitory, are due to a lowering of functional activity in a distinct part of the speech mechanism caused by the upsetting of balance between the several parts of the mechanism produced by the destruction of one of the integral parts by the lesion. He insists that the nervous system acts as a whole and so with the speech mechanism. That is, if in a case of motor aphasia, Broca's area is destroyed, the independent areas which von Monakow believes exist, according to the old diagrammatical scheme of localization, also suffer, and so a lesion in the motor speech area would produce

a depression of function in the visual, auditory and other speech areas and thus cause the resultant pronounced aphasia. Von Monakow believes with Marie²², Grosset and others that the aphasia is simply a defect of intelligence as originally held by Trousseau. In fact, Marie lays great stress upon the intellectual defects shown by patients with aphasia and insists that those who deny this viewpoint have neglected to use even the simplest of intelligence tests. He states that he has been able to prove beyond doubt the obvious lowering of the intellectual level in every case of aphasia under his observation. This author cites numerous cases, not of speech intelligence loss, but of actual defects of general intelligence which can be found in every aphasic. We have as illustrative of this point the following clinical cases:

M. A., female, aged 33 years, Italian. Married, has two children. On August 31, 1917, while at home at her work of dressmaking, she suddenly found that she had lost her power of speech. She did not fall and was assisted to bed. There was no weakness of the extremities but her mouth twitched and was drawn towards the left. She slept for three or four hours, then arose and found that she could say only "Dio" and "che cosa." Her husband asked her whether she knew him. She shook her head and could not respond.

She came to the Vanderbilt Clinic September 19, 1917, and it was thought that she might have a psychosis of some kind. On account of her inability to speak English she was turned over to me and after due study, the proper diagnosis was made. The only point of importance in the history is that she has suffered from valvular heart disease for ten years and has had to have two induced abortions on account of this condition. She had felt perfectly well mentally before the onset of her illness excepting that her heart continued to give her trouble. Since her illness her mood has been depressed; she seems to be bewildered, dull and indifferent, will sit for hours doing nothing, and will not speak unless spoken to. Her thought content is hypochondriacal. She says that she is never going to get well, that she is unhappy and wants to die, and she refers

frequently to her desires about returning home to her parents and children in Italy. She repeats continuously: "Here in this country there is plenty, but there in Italy my parents and children are starving." She will not, or cannot, do any housework but eats very well and sleeps uninterruptedly.

The aphasic status taken on September 21st, is as follows:

"Io mi chiamo Ansaldi Marguerite e son de trenta-tre anni il mio marito lavoro come cuoco nella cuilina di un hotel Americano."

Answer: "Io mi chiami Anan ansaldi margurtr e nari di—" A hopeless jargon—does not understand what she reads at all. Cannot copy from writing.

Question: "How many letters are in the word 'Presidente'?" She answers 9, and is not able to answer at all unless she is allowed to look at the word. Asked to write what is the trouble with her, she writes jargon: "Silisena lare serlal nesse el nosilanesesto nelano—" (Jargon).

Asked to subtract 7 from 10, she answers 6, even when shown the problem on paper. Asked to subtract 5 from 10, she says 5, but when asked if she is sure she hesitates and says "I do not know." Asked to write the days of the week, she writes as follows: "selene, nestani, nerten, lenerti, selani, enonenito—" (Writes all words wrong).

October 19, 1917.

Question: "What is your trouble?" Answer: "I feel weak in the heart. I can't read much and do not understand what I read." Counts fingers and gives the days of the week. Reads mechanically and with many mistakes. Does not understand what she reads and is not able at all to give in her own words the substance of any written or spoken paragraph. Cannot write spontaneously, and makes many mistakes in writing from dictation and from copy. She hears and understands a great deal of what is told her but often makes a mistake and is not able to correct it. Has to be told several times to get up and open the door, to sit down, etc., before she understands. Lichtheim's test is well done. She can tell the number of letters in the word but occasionally will fall down on the syllables, although she insists that she knows what syllables are. Grammatical mis-

takes are made only in writing or reading, very rarely in speaking spontaneously. She remembers and names objects. There are alexia and agraphia.

November 28, 1917.

She is asked to read the headlines in an Italian paper which show that the Italians are fighting bravely on the Piave line and are holding the Brenta sector although they are outnumbered five to one. The article deals with the heroic resistance that the Italians are making successfully against their enemies. In asking her, first, to speak spontaneously her conception of what has been read to her, she gives a satisfactory, although a very meagre abstract, but in writing, although I waited patiently for her to answer for more than a half hour, she never got further than the phrase "Gli Italiano," which is incorrectly spelled and means The Italians. She said that she was able to understand thoroughly what she wanted to say and she made the motions of writing perfectly, as can be seen in her broken phrase just mentioned, but found it absolutely impossible to reproduce what she wanted to say. This, despite the fact that there was no paralysis in any part of the patient's body at any time during the long period she was under observation. It is truly remarkable the extent of the damage which was done to this patient's brain without producing paralysis. She continues to improve in her spontaneous speech, making fewer errors, but occasionally finding it very difficult to pick out the word. On such occasions she often substitutes an irrelevant word or phrase for the one she wishes to use. A steady improvement is taking place which is most noticeable in her speech and in reading. She reads a good deal better but still makes a great number of mistakes which, however, she occasionally recognizes. She makes but poor progress in writing and still is as bad as she ever was.

A.M., male, aged 19 years, single. Italian.

The patient had bronchopneumonia three years ago. The left iris was operated on for cataract when he was a baby and on three subsequent occasions. Aside from these two incidents, the patient had been perfectly well until April 14, 1917, when he was

operated on for appendicitis at St. Catherine's Hospital, Brooklyn. Three days later he became paralyzed on the right side of the face, in the arm and leg, and became unable to speak. About two years ago, the boy is said to have had an epileptic seizure. The attack was very severe and was repeated about one year ago. These two epileptiform convulsions were not followed by speech defects or by paralysis, and have not been repeated to date.

The patient is the eldest of eight children; all of the seven are well and the father and mother are both living and well. The mother had two non-induced miscarriages.

In order to understand the aphasic status of this patient, it is necessary to know that he went to school at six, attending the public school until he was 16 years of age. When he left he was in 7-A, and, although 16 years of age, he did not graduate and would not have graduated until six months later had he stayed in school. He was not promoted on three occasions, partly because he did not attend regularly, and partly because of some trouble with his left eye, which was operated on and kept him out of school at various times almost continuously for three or four months. Aside from this, however, the boy was a truant and was unmanageable in so far as his attention to school duties was concerned. He was always bright and keen and picked things up easily, read a great deal of boys' fiction and newspapers, but nothing serious. He wrote a fine hand and was able to compose a fair letter to his friends and relatives. He spoke Italian fairly well, but English was always the language of choice and was spoken much better than Italian. He never could write Italian and, of course, cannot do so now. In fact, this boy can be said to have had the average education of a boy who leaves school in the grade known as 7-A. The average age of these children is 12 years. It will be remembered that he was 16 when he left.

Four years ago he began to work in a shoe factory where he stayed for three months and then went to a marble works for seven months, and back to the shoe factory until recently. This patient, and also the one above reported, are right-handed, and have come from right-handed families.

The aphasic status, following the order of our fifteen questions, is as follows:

1. The patient cannot speak spontaneously in ordinary conversation.

2. Can be done with a defect. The defect appears to be mostly in naming objects.

3. He cannot speak from dictation.

4. He cannot read aloud.

5. He can read to himself and seems to understand what he reads.

6. The patient cannot repeat aloud what he has read, cannot say his prayers aloud, but he can sing "My Country, 'Tis of Thee" all through without a mistake in the words, but when asked to repeat the words of the song, he cannot do it.

7. He can write spontaneously but uses wrong words. For instance, when asked the question, "Please write down what troubles you have," he puts down the phrase, "I am England." When asked what this means, he replies, "I had appendicitis." When asked to write my name, he wrote, "Dr. Frisco," Teddy Roosevelt, was written "Tippery."

8. The patient can write the numbers from 1 to 10. He can add up simple columns of figures and can write the days of the week and can also write the names of various objects which I showed him, but will often make mistakes which he frequently corrects himself.

9. The patient can write only simple things from dictation, but leaves out letters and quite often words, and in Question 10, he will do the same thing, i.e., he can copy from print but will omit words or letters.

11. The patient hears and understands what he hears.

12. He sees and understands what he sees. A peculiar reaction which should be noted is one in which I asked him to look at my watch and tell me what time it was. He could not do so in words, but he drew a diagram of a watch with 13 numbers on it, there being 2 tens, and then drew the hands of the watch on the blackboard, so that the result of the diagram presented the correct time, which happened to be five minutes to four.

13. He is aware of errors in both speaking and writing, but is not able to correct them.

14. The patient is able to tell the number of letters in a word, but does not seem able to

tell the number of syllables, although he assures me he understands what syllables are. July 2—After a long explanation he finally demonstrated that Lichtheim's test is positive. He was able to pick out the number of letters and syllables in many words.

15. The patient is practically wordless. Therefore, no ungrammatical uses of words could be made out. Whatever utterances he has, and they are very few indeed, he perseverates.

HISTORY AFTER THE ONSET.—This patient at first was able to say only "Yes" or "No." Soon thereafter, however, he began to say words, but spoke a hopeless jargon and was only able to give his own name and that of his father and several of his relatives. He used "Yes" and "No" properly for assent and dissent, but aside from this could not be said to have very much spontaneous speech. He has always had the power of mimicry very highly developed and can make himself understood by various means in this way. He has learned to use his left hand very quickly in writing and does fairly well. He has improved a great deal, particularly on the sensory side, being able to understand a great many more things that he sees and hears than he did at first. He still speaks and writes a hopeless jargon, however.

This patient indulges now (9 months after onset) in much silly behavior, and is the butt of a great deal of ridicule at the hands of other clinic patients because he spends most of his time trying to use his charms on the clinic nurse. He comes to see her a great many days when it is not necessary for him to attend the clinic and has made himself so ridiculous in his attentions to her that other patients laugh at him. His grasp on school knowledge and current events is poor and he indulges in much foolish rhyming and grimacing.

J.M., male, 36 years of age, married. Irish.

HISTORY.—On Friday, December 16, 1916, this patient went to sleep at night apparently perfectly well and woke up the following morning complaining of weakness in the right arm. There had been prior to this no headache, no dizziness or numbness, and the weak-

ness in the right arm was not accompanied by any of these symptoms. Later in the day the patient became drowsy and finally unconscious. His wife in the meantime came home from a shopping trip and found him on the floor paralyzed on his right side. His eyes were open and he seemed to recognize her, but could not speak. She states that she had been gone not more than one hour and that when she left he was not unconscious. The patient says that he was unconscious during this hour that his wife was gone, but we have no means of being absolutely sure of this point. We are certain, however, that he was not unconscious more than one hour, if at all.

PREVIOUS HISTORY.—On March 19, 1916, our patient was struck by a street car over the right ear, contusing it and wounding it severely; the right side of the face was also contused. Immediately after this injury he complained of deafness in both ears, worse on the right side. Following the injury, he did not bleed from the nose, from the ears or into the eyes, and is said at the hospital where he was observed not to have fractured his skull.

The patient has led a very active life, has been a soldier in the English army, and has been all over the world, most of the time in tropical climates. He has been admittedly a very hard drinker. Venereal infection is denied.

In order to understand the aphasic status in this case, it is necessary to know that he has been practically illiterate, that he could read very little, and could write less, before the onset of his trouble. He was able to read simple articles in the daily newspapers with difficulty and could only write the simplest of letters. The deformity of the right pupil is congenital and has been diagnosed as a cataract. The family history is negative, the patient not knowing the cause of death of his parents and other relatives.

1. The patient cannot answer this question.
2. These questions were answered well although there is a slight defect in the naming of the members of his family and the days of the week.

The patient failed in answering the 3rd, 4th and 5th questions.

In the 6th, he failed also. Although he can

carry a tune perfectly well and gets many phrases in the song, he cannot be said to sing the words intelligibly.

In answer to question 7, the only thing the patient can do is to write his own name, and very frequently only his last name.

8. The patient can write from 1 to 10, but cannot add, cannot write the days of the week and cannot write the names of objects shown to him.

9. He cannot write from dictation. In considering these last two tests, it must be remembered that this patient is possessed of a very poor quantity and quality of education and some allowance must be made for this lack.

10. The patient practically fails in doing this test, but can copy his own name and his wife's name and very little else.

11. He hears and understands what he hears perfectly. He recognizes the watch when placed close to the left ear, the right one being deaf. The patient's eyes were closed in the watch test. He obeys very well all spoken commands, provided they are not too complicated.

12. He recognizes everything that he sees; his answers are all correct.

13. He is well aware that he makes mistakes in speaking and writing.

14. The patient's idea of his difficulty is that he understands everything and has the mental picture of what he wants to say but cannot say it. Lichtheim's test could not be made on this patient because of the lack of education. He does not know what syllables are and, therefore, naturally could not be expected to perform this test properly, although he is able to tell the number of letters in most words, particularly words and names with which he was previously familiar before the onset of his trouble. If this question must be answered, we should have to say that the patient has a correct conception of his speech.

15. The patient occasionally misuses what few words he does utter, speaks a jargon and is prone to repeat.

This patient is subject to quick changes in mood, shows great lack of interest, drinks a good deal and is very untidy and careless about personal habits. He is bad tempered at times but usually smiles and shows an utter disregard for the seriousness of his condition.

M. G., female, 20 years of age, married, 1 child. Italian.

FAMILY HISTORY.—The patient's father and mother are living at 45 and 39 respectively. The father is very alcoholic and beats his wife and children, especially when he is drunk. There are seven brothers and sisters, all living and well. The patient's child seems to be normal. The husband denied any venereal infection whatsoever. The patient has been known to her husband for 19 years, and while she may never have been very bright or intelligent, she was considered a normal girl of her class and education. She had a normal, jolly and open disposition and there are no seclusive symptoms in her make-up.

HISTORY OF THE ONSET.—On June 11, 1917, the pregnancy terminated and labor began. Several hours afterward the attending physician insisted on a consultant, because forceps were deemed necessary. While she was under anæsthesia, two doctors extracted the child and after the labor both mother and child were apparently in normal condition. Suddenly, two and one-half hours later, the patient was seized with a generalized convulsion, particulars about which are impossible to obtain. For instance it is impossible for us to know whether the convulsions consisted of generalized tonic or clonic spasms, or were limited to twitchings to one side. It is said that the patient was unconscious during this convulsion, which lasted about one-half hour or more. A second convulsion seized her five hours later, and during the night several others; altogether the patient is said to have had five convulsions. She was practically unconscious for three weeks and during this time, had one or two more convulsions.

She was treated during her unconscious period for eclampsia, and in applying hot water bags for the relief of the supposed kidney condition, horrible burns of a second degree were inflicted upon the patient practically over the whole back. At the end of three weeks, when the patient regained consciousness, it was found that she was paralyzed on the right side, including the face and both extremities. It was found, also, that the patient was prac-

tically wordless, her utterances being limited to the words "Yes" and "No," which were not always used properly, as a means of assent and dissent. She did not seem to understand what was asked her, and it was considered that she had suddenly become demented. She was taken to St. Michael's Hospital in Newark and remained there for six weeks. By this time the paralysis on the right side of her body had cleared up considerably, being reduced to a hemiparesis. The speech condition, however, was practically not at all influenced, and the patient's condition has continued as it now presents itself since then. The aphasic status is as follows:

There is no answer at all to Questions 1, 2, and 3.

There is complete failure in answering Questions 4, 5, and 6.

7. She can only write her first name "Mary" and makes the first letter of her second name (maiden name) which is T., but goes no further.

Complete failure in answering Questions 8 and 9.

10. She is only able to copy her written name. This should be considered a complete failure.

11. Patient does not stand up when asked to do so. When a watch is held to her ear, a look of intelligence and recognition spreads over her face, but she is not able to tell what she hears or describe, write or indicate in any manner the name or use of the object.

Complete failure for Questions 12, 13, and 14.

15. The patient is wordless except for the words "No," "Yes" and "All right," which are not always correctly used.

It must be remembered that this patient had an ordinary public school education, that she was able to read and write commensurately with one in her position, both in Italian and English, but now can do neither.

This patient was sent to me for commitment to a hospital for the insane, it being considered she was demented. There is no case that I can remember to have seen or read of which can approach the depths of loss of all intelligent speech function which was reached in this case. She had absolute aphasia and apraxia. She did nothing purposeful with either extremity of both sides, in the absence of sensory

or motor paralysis and ataxia. She could obey no command, either by the aid of mimicry or imitation, or in any other manner. The simplest and most primitive suggestions given in various ways were not perceived. She simply laughed immoderately, and if she thought the examiner was displeased, would cower in fear or would attempt to placate by guttural and throaty sounds of pleading.

While the views of Wilson are very clearly set forth, he seems to have followed other workers in the field of aphasia and apraxia in so far as he has also made reference to various claims of localization, and quotes diagrams purporting to explain the mechanism of the production of apraxia and its localization. As previously with aphasia, the statement is again made in the case of apraxia, that in right-handed individuals the lesions causing both of these conditions are to be found in the left hemisphere. This writer carries the analogy further by recognizing also in apraxia, as in aphasia, cortical, subcortical and transcortical apraxias. A great point is made of the fact that apraxia is always supracapsular and may exist without any sort of paralysis at all, the capsule escaping responsibility in all cases. Wilson draws a close analogy between motor aphasia and apraxia. Indeed, he says that "in motor aphasia we have a form of apraxia of the speech musculature." He ascribes the loss of motor speech to the inability to articulate auditory, visual, and, most important, kinesthetic word images. Agraphia and the instrumental type of amusia are also special kinds of apraxia, and he ascribes the loss of function in these cases to lesions of the centers for graphic kinesthetic images, or the visual image of writing, or to a loss in the image of the movement for writing or playing instruments. In other words, his ideas of apraxia follow very closely the various theories of localization as set forth by Broca, Wernicke and Villiger in aphasia, and these theories are absolutely dependent on the conception of more or less circumscribed centers connected in each hemisphere and interconnected between hemispheres for the production of voluntary motion. This, in spite of the fact that it has been impossible to show by anatomical or pathological investigation that any such centers exist either in apraxia or aphasia.

Wilson, however, restricts the term apraxia to the motor side, following Liepmann in this respect, for he says in the same article that "in regard to the lesions which underlie ideational agnosia, or ideational apraxia, for that matter, there is little to be said that is definite." In this connection, the pathological anatomy of the subject of apraxia will show by reference to the literature of cases reported by Stransky³², Marcuse,²¹ and Pick²⁴ that in a great many of these cases the lesions are too widespread to be of any localizing importance. Only in the matter of motor apraxia does there seem to be any ground for the belief that the supra-marginal and angular gyri and the first and second left frontal convolutions have anything to do with this difficulty.

The original case of apraxia described by Liepmann¹⁹ was one of motor aphasia and right-sided apraxia. At postmortem, it was found that the whole corpus callosum was destroyed excepting the splenium, that there were two foci of softening in the white matter beneath the first and second convolutions of the left hemisphere, also that there was a large sub-cortical cyst beginning under the lower third of the postcentral gyrus, spreading back through the parietal lobe beneath the supra-marginal gyrus, and ending at the posterior part of the angular gyrus. Besides these lesions in the left hemisphere and in the corpus callosum, there was a focus of softening in the right hemisphere and in the internal capsule, and another in the neighborhood of the supra-marginal and angular gyri. So that this case, which formed the basis for the original conceptions of Liepmann of motor apraxia, is very unsatisfactory from the viewpoint of pathological anatomy in relation to localization. Most patients suffering from apraxia have arteriosclerosis. Von Monakow³⁴ declares that generalized cerebral vascular sclerosis plays an important part in interrupting association tracts and also in causing deficient nutrition to all parts of the cortex. So that areas of softening in any one of several spots, due to vascular change in the vessels of the brain, may not entirely explain the symptoms; for it is inconceivable that the defective nutrition caused by the arterial changes in all parts of the cortex distant from the lesions found, may

not also help to explain some of the trouble. For this reason, a statement by Liepmann in regard to apraxia and by others regarding aphasia, that these conditions are more common in lesions of the left hemisphere, is not borne out by pathological investigations, because single lesions in either condition are common and seldom are the lesions found limited to either hemisphere alone. Clinically, however, the statistics of Liepmann seem to confound this statement. These statistics show:

Of 89 patients, 42 had left hemiplegia and 41 had right hemiplegia, 5 had aphasia but no hemiplegia and one was purely apraxic only. In describing the left hemiplegias, Liepmann says that nearly all were able to execute given movements with the right non-paralyzed arm. Of 41 right hemiplegics, 20 were apraxic and there were 14 also aphasic, which demonstrates rather conclusively the intimate relationship between these two conditions, although these figures also demonstrate that the conditions are not necessarily associated. It seems rather to be deplored that most of the cases of apraxia which have been investigated with pathological findings have been patients who were either old dementias of senile or arteriosclerotic origin or sufferers from alcoholic dementia. This is especially true of the work of Bonhoeffer¹ and Pick²⁵. While it seems to have been fairly definitely proved that the great majority of cases of aphasia and apraxia occur in left hemisphere lesions in right-handed individuals, it has not been so definitely proved that the remaining cases of aphasia and apraxia necessarily occurred in individuals who were left-handed, and who, therefore, may have had a specially developed right hemisphere. We shall be able to show several cases which will clinically illustrate this point.

It is not absolutely essential to our conception of speech disturbances or voluntary movements that they be localized in either hemisphere alone, and it would seem that caution should be used in adhering strictly to the viewpoint that these special skilled acts, of which speech is one, have their correlating centers almost exclusively in the left hemisphere. In nearly all of the reported cases of apraxia with autopsy, and in a great many cases of aphasia with autopsy, lesions are found

in both hemispheres. It may be assumed with fairness that various individuals in attempting to fit the findings with their theories have unduly emphasized the findings in the left hemisphere. It seems that the most sensible view to take of the entire matter is that the whole cortex is indivisibly associated with the development of the function of speech and its expression, and also with the development of the function of specialized purposeful movement and its performance. True, in certain individuals, the left hemisphere acts as the leading or dominating organ for the inception of these functions; but in a great many cases, the conditions may be reversed. In this connection, Liepmann has always denied that either one of the hemispheres is necessarily the dominant factor in the production of apraxia. He also denies the existence of an apraxia center in either the supramarginal or angular gyrus. His contention is that any such center or group of centers cannot exist independently of the motor sensory area grouped around the fissure of Rolando. The main point in this discussion for him is the function of the corpus callosum, particularly that portion through which fibers come from the anterior central convolutions, namely, the middle third of the corpus callosum. In illustrating this point, Liepmann calls attention to the fact that in most cases of apraxia due to lesions of the corpus callosum, the posterior 4th or 5th is intact. Secondly, cases have been repeatedly observed in which the anterior portion was destroyed without apraxia being present, and thirdly, there is a case on record in which apraxia has occurred when the anterior portion of the corpus callosum was entirely intact.

Bearing on the point of stock-brainedness, that is, the tendency in families to have the left or the right hemisphere predominating for speech and voluntary movement, as is shown by the right- or left-handedness of such individuals, the work of Kennedy¹⁴ may be of interest. Before referring to this author, it might be well to state that according to Liepmann's researches, 95½ per cent of all individuals are right-handed, and, therefore, their left hemispheres lead in the production of purposeful movements and in the utterance of speech. Kennedy asserts that while certain

individuals of a right-handed stock may be left-handed, in these individuals the speech center is always in the left hemisphere. Kennedy also applies the same rule to individuals of left-handed stock who are right-handed, the leading hemisphere in such individuals being the right one. He calls on these observations to explain the existence of crossed aphasia, that is, aphasia occurring in right-handed individuals with left hemiplegia and in left-handed individuals with right hemiplegia. The facts of stock-brainedness are purely speculative and have never really been proved by any extensive statistics; nevertheless, Kennedy reports seven cases as a basis of his conclusions, which it might be worth while to set down in short abstract form.

The first was a French sailor who suffered a very extensive destruction of the right hemisphere with a resulting left hemiplegia, hemianesthesia and hemianopsia. This sailor was left-handed in everything but writing, which he was able to do, however, with either hand. Although the lesions in the right hemisphere were very extensive, there was no evidence of aphasia at any time. Kennedy states that this man was the only member of his family who was left-handed. Nevertheless, the fact that he was able to write with his right hand is overlooked, and also the further fact that his ambidextrous accomplishments may have been acquired, and that, after all, the left hemisphere may have been the leading one, despite the author's conception.

A second case is a young man of 36 who had a right-sided hemiplegia and was aphasic. He was always left-handed except in writing, and was the only member of his family who could use his left hand for skilled acts. Here the same remarks apply as in the foregoing case.

A third case was a left-handed individual who developed a left hemiplegia after a fracture of the right occipito-temporal skull and middle meningeal hemorrhage. He was the only left-handed member of his family. No aphasia developed. Again, the remarks made in the first case are pertinent.

The fourth case is a right-handed girl who had severe left-sided attacks of epilepsy followed by severe but transient hemiplegia and aphasia. This girl's mother and father were

left-handed and the inference is that her speech centers were in the right hemisphere which, according to Kennedy's theory, should also have been the leading hemisphere for all sorts of skilled movements. Whether these cases are illustrations of the theory of stock-brainedness or whether they are merely examples of the point Hughlings-Jackson so often made, that no particular hemisphere has the exclusive function of the speech mechanism, is a salient point.

The fifth case occurred in a young man who suffered from complete aphasia for ten days following a right subtemporal decompression for a right-sided subcortical Rolandic glioma. This patient was right-handed, but two brothers were left-handed, and it is presumable that Kennedy wished to imply that in this individual the right hemisphere was the leading one for speech.

The sixth case was a woman of 67 who, following a cerebral hemorrhage, had a left hemiplegia with mixed aphasia. She was right-handed, but her mother, brother and a daughter were left-handed; so that presumably the right hemisphere in this individual was the leading one for speech.

The seventh case was one of left hemiparesis, hemianæsthesia and hemianopsia with no aphasia. He had been left-handed all his life and was ambidextrous in regard to writing. The rest of his family were right-handed and so presumably the left hemisphere should have been dominant for speech in this individual.

The fallacy of this sort of observation can readily be seen when attention is called to the fact that a great many cases of right hemiplegia occur without any sort of speech defect whatsoever, regardless of whether the individual is right-handed or not. We have all seen this a number of times. Some support is given, clinically at least, to Jackson's conception that it is not essential for either hemisphere to be the dominant one for the production of speech or purposeful voluntary movements. The following cases will tend to illustrate, clinically, the difficulty and the dangers of ascribing too much importance to the accurate localization of the speech mechanism in either hemisphere alone:

E. C., male, 51 years of age, single.

HISTORY.—He was taken on the 14th of November, 1917, at 10:30 A.M. from a saloon by an ambulance surgeon from the Kings County Hospital. While standing at the bar he had suddenly become unconscious and had fallen to the ground. He had had no prodromal signs of any description. When he was brought to the hospital, he was unconscious, temperature 99, pulse 96, and respiration 18. He remained unconscious for two days and the blood count and urine taken immediately were negative. The blood pressure was 125 systolic and 70 diastolic. The outlines of the discs were blurred and showed some edema and the vessels were congested. He had no convulsions during this time, and at the end of the second day he could be aroused only with extreme difficulty, would open his eyes, but could not speak a word. He moved his right arm and leg, but the left arm and leg were completely paralyzed and flaccid. The Marie-Foix sign was present on the left side and there was an Oppenheim and Babinski sign on the left side with clonus. The left abdominals were gone and the left side of the face was paralyzed. The tongue deviated to the left and the uvula somewhat to the right. The pupils were normal in every respect although somewhat dilated.

On the evening of the second day he had a nosebleed and the sight of the blood started him speaking, indistinctly however. He spoke a senseless jargon which could not be understood at all. A Wassermann taken of the blood at this time came back negative. This was repeated three times and found negative each time. There is, however, the history of a chance twelve years ago with treatment at Hot Springs by mercury and inunctions, for a period of about two months. Further questioning brings out the fact that he had an attack of palsy on the right side of the body three years ago from which he recovered entirely at the end of two weeks, having had at no time any speech defect whatsoever, although the attack was initiated by a period of unconsciousness lasting several hours.

This patient played ball and did his mechanical work always with his left hand and favored his left hand in doing all sorts of skilled acts,

except writing. He always wrote with his right hand and wrote a very fine hand. Spinal puncture was denied.

When the patient said his first words at the end of the second day he could not make himself understood at all. A day or two later he could use "Yes" and "No" for assent and dissent and could give his name. Shortly after this he tried to give a description of his accident in halting, stumbling phrases with great difficulty and made many mistakes which he, however, immediately recognized and tried to correct. He wrote a good account of his whole trouble without difficulty; heard and understood what he heard, saw and understood what he saw, and did Lichtheim's test well, both for letters and syllables. He could read spontaneously and from dictation, understood what he wrote and could write from copy. He understood perfectly what was read to him or what he himself read, but became lost in a meaningless jumble of phrases when he attempted to speak. He was very much excited and very much upset. He was encouraged to write and to read aloud, and the improvement in him grew quite rapidly, so that at the end of a week he could make himself understood although his vocabulary was still quite limited.

On November 27, two weeks after the accident, he had, to all intents and purposes, recovered entirely. He could read and speak well, and the paralysis on the left side had improved synchronously with the improvement in speech. The physical signs on the left side had disappeared entirely, and the patient was discharged apparently perfectly well.

R. B., male, aged 29, married.

He came to the Vanderbilt Clinic May 11, 1917, complaining of paralysis of the left side of the body, including the face, arm and leg, the loss of memory, and, as his wife expressed it, "foolishness." This condition had lasted three months and has remained stationary.

HISTORY.—On December 17, 1915, one and a half years before the onset of his present trouble, he was hit by an automobile in the lower spine. He was thrown to the ground and was unconscious for two hours. He was taken to Bellevue Hospital where an x-ray and blood

examination showed nothing in the way of fracture, dislocation, or disease. He was discharged from the hospital with a diagnosis of contusion of the spine, and was returned to his home practically well, excepting for a soreness in the lower dorsal region when he bent forward. This pain in the back persisted for several months and finally disappeared. For two months before the actual onset of this trouble, the patient complained of headaches and vomiting nearly every day, usually at night after his supper. During this time, too, his general disposition seemed to undergo a change. He was unusually irritable and restless, and gave way to explosive outbursts of temper. He did not complain at any time of dizziness, nor did he have perversions or subjective changes of sensation in any of his limbs. There was no numbness or weakness and never any disturbance of speech during this prodromal period of two months before the onset of his aphasic condition.

The patient's wife says that he has never been ill, and he says he has never suffered from any venereal infection and that for a number of years he had been in the habit of drinking about one pint of beer a day. They have had two children, both of whom are well. The wife never had any miscarriages.

It is well to note the early history of this patient. He was born in New York City, began school at the age of six and graduated at the age of 14. He was bright and was promoted regularly, but was never very fond of attending school. After graduating he went to La Salle Academy for one year and quit soon after his father died in order to go to work. The only foreign language which he ever tried to study was French for one year at La Salle Academy. It cannot be said that he ever really knew any French, and he forgot what he did know long before the onset of his trouble. He was always considered a fairly good singer. He never had any musical education, either vocal or instrumental. He was always rather wild, and preferred to dance and play pool rather than study while he was at high school. He always preferred mechanical work to study of any kind. He never read anything except newspapers, and can be said to have had only an ordinary public school education. Even

mechanically, however, he was never very skilled and was only a laborer in the highways department of this city when attacked by his present trouble. He and all his family are right-handed.

ONSET.—This patient was in the habit of going to work at night as a watchman. At 1 o'clock his actual work was finished and from then until 7 o'clock in the morning it was his custom to sit on a chair or pass the time as best he could. It is definitely known that until 1 o'clock on the morning of February 25, 1917, he had been in his usual health; in fact, for a few days before the onset he had had no headaches, no vomiting. Nothing is known of what occurred to him after 1 o'clock on this particular night, and he himself is not able to explain, excepting that he sat down to rest and to sleep until morning. He was not seen again and nothing is definitely known about him until a chauffeur found him unconscious on the floor about 7:30 A.M. It is known positively that he was unconscious for an hour thereafter. When he regained consciousness he did not speak for more than a week. He was absolutely wordless. During the second week he began to call his wife by name (May), then his boy Willie, and finally his sister Hannah. He could say "Yes" and "No," but did not always use the words properly; and he could ask for a drink. During the second week he was very bad tempered. He remained in Bellevue Hospital for three weeks and during this period he could say no more than what has just been mentioned. He was five weeks in the City Hospital and here he improved physically and was able to get up and walk, but mentally he has remained practically the same as now. He has added but very little to his speech and the condition has practically continued the same since the onset.

The aphasic status, in the order of our fifteen questions, is as follows:

1. "Cold—none—stroke—speech." "I do not know." In other words, there is no spontaneous speech.

2. Counts all fingers, then says "Two." Asked how many fingers on both hands, says "Four." "Willie, Annie," says "Maggie" then

"May," and then mentions the name of his niece, "Genevieve." He was asked to limit his answer to the members of his immediate family. He included his sister and niece (Hannah and Genevieve), and made a mistake at first in mentioning his wife's name, so that we may consider this a defective answer.

Asked to name the days of the week, he said "August" and the second time "September," which, of course, makes this an absolute failure.

Asked what an inkwell was, he responded "ink." Pointing to a yellow sheet of paper and asking for the color and the name of the page, he answers, "paper." Pointing to a magazine, his answer is "book." Pointing to a rubber stopper in a bottle, his answer is "celluloid." Pointing to a blotter, he says "boiler."

When asked to name the number of chairs in the room, he gets up to close the door. When asked the color of his tie, he says "blue," then says, "I don't know." (The color of his tie was black.)

3. He cannot speak from dictation.

4. He cannot read aloud what is printed.

5. When asked to read to himself from a newspaper article, he gets up to look for a piece of chalk and finally asks for the chalk by name. The only thing which he can read is his own name, that of his wife and child and very little else. He does not understand what he reads, even though the article is a simple one.

6. He cannot recite his prayers, with which he was previously very familiar. Cannot sing his favorite song, which was "River Shannon." Cannot even hum it or whistle it tunelessly. Tone and cadence are missing from the whistling and humming.

7. He cannot write spontaneously except his own name and his wife's name. He occasionally attempts to write to friends and the result is a hopeless jumble and jargon. It is interesting to note that when he wrote to his wife from the hospital he always addressed her by her maiden name.

8. Writes from 1 to 10. He cannot write the days of the week nor the date. Does incorrectly even simple sums. Cannot subtract and cannot multiply or divide.

9. Cannot write from dictation.

10. Cannot copy printing or writing. When asked to copy a simple sentence from a magazine he answers by writing his name.

11. He hears but does not understand what he hears.

12. He sees but does not understand what he sees.

13. He is apparently not aware of errors made in writing or speaking. He seems unable to say definitely whether he makes mistakes or not. Certainly he does not correct them when made.

14. He appears to have no conception of his speech as tested by Lichtheim's test. He cannot tell how many syllables or letters there are, by any means of indication, in any words, not even his name. Occasionally he can tell the number of letters in his name and in the name of the president of the United States, but he is not always correct. He has at no time been able to tell us the number of syllables in any given word correctly several times in succession.

15. He misuses words; he is ungrammatical out of proportion to his education. He repeats fixed phrases and when he does speak or write, it is a hopeless jargon.

The family history in this case is very interesting. His father is dead, cause not known. The mother died at the age of 43 from an attack of apoplexy, after an illness lasting only three days. The onset in the mother's case was very sudden and she never regained consciousness, so that the supposition is that she had a cerebral hemorrhage. The rest of the family consists of two sisters. One of them is perfectly well physically and mentally. The other, aged 32, had a right-sided apoplectic seizure two years ago without a defect in speech of any kind whatsoever. She did not make a good recovery from this, although she improved somewhat until three weeks ago, when she had a second similar right-sided attack of hemiplegia. The Wassermann is negative in both the sister and patient, the patient having had numerous spinal fluid and blood Wassermanns performed at Bellevue Hospital and here, all of them negative.

The physical examination shows the usual symptoms of a profound left hemiplegia.

CHAPTER IV

The conceptions of the development of speech and the mechanism of its production are intimately bound up with the problem of the cause of voluntary movements and the development of purposeful skilled acts. Psychologists have taught, and clinicians have followed their lead in teaching, that the idea of a movement, whether it be the idea of a movement of speech or any of the other purposeful movements, exists in consciousness and precedes the execution of the act. This is particularly brought out by William James¹³ whose conception is that among the sensations produced by a movement there are some which are caused by stimulation of sense organs not located in the moving members. These sensations are called by James remote sensations, and they are not the constant and variable results of the movement. For instance, "the eyes may close or turn to one side and the ears may be filled with the noise of other things." But what he calls resident sensations are the muscular and cutaneous sensations which originate in the moving member and are constantly necessary for a given movement. It is these kinesthetic sensations, according to the psychologists, which give us the knowledge of our movements and constitute the ideas of movement. In other words, to will a movement is to bring into consciousness the kinesthetic memories of such a movement. The idea of the movement must be present before the will can work on it and the voluntary act follow. Therefore, given the kinesthetic or sensory image of the movement and how it is going to feel, the next step, according to this psychological conception, is the performance of the actual movement itself. This idea is brought out in the words of William MacDougall:²⁰ "The kind of idea that tends to issue most directly in action is the idea of a movement, the kinesthetic idea." This is illustrated by the author in the case of the beginner in golf who is coached in addressing the ball by an expert. We quote: "The expert commands a readjustment of this and that limb, of the trunk and head, until the proper attitude is struck, and it is the learner's task to combine the kinesthetic impressions which

NOTE: In both of these cases the ascendants were right-handed.

this attitude yields to a single percept that can be produced on future occasions. Frequent repetition of such a series of movements under similar conditions results in their becoming what is called secondarily automatic."

Many authors, particularly Liepmann, have persisted in placing this area for the reception of kinesthetic impressions in the motor cortex and in connection with speech production; others, including Déjerine, as we have already seen, have placed the area for the reception of the kinesthetic images of speech in the left third frontal convolution. We hope we have, however, succeeded in throwing a great deal of doubt on the proposition that this area of the brain possesses this function, as applied particularly to speech. We have adduced considerable pathological and anatomical proof against this conception in the work of Marie, Moutier and others already quoted. Psychologically, the conceptions of MacDougall and other psychologists have been disproved by Woodworth.³⁶ In his experiments, the subjects used were young adults, two women and eleven men, nearly all of them persons possessed of psychological training. The experiment consisted in requesting these individuals "to make given movements with some preliminary hesitation, and to note the condition of mind that preceded the movement. He was to note particularly what imagery appeared; and in the case of motor images he was asked to compare them with the sensations resulting immediately afterwards from the actual movement." Stress was laid on the necessity for the slow performance of movements so that imagery would be given more time to appear. Prompt movements were also made which, however, were less often accompanied by any sort of imagery. The movements consisted of opening the mouth, wagging the jaw, winking and closing the eyes, flexing or supporting the fingers, flexing the foot, opening and closing scissors, forceps or manipulation of the dynamometer. In some cases a choice of various movements was allowed; for instance, the hand was to touch any part of the body or an object in the foreground. The fingers were to be flexed or extended, or a reaction was to be made to a sound with either the hand or the foot.

"The results of 128 single introspections of

the conscious preliminaries of voluntary movements showed:

27 gave kinesthetic imagery.

27 gave visual imagery.

17 gave imagery of other kinds.

30 gave only peripheral sensations.

27 gave an absence from the field of attention of all sensorial elements, whether external impressions or images.

"In 55 per cent some sort of imagery occurred, but kinesthetic imagery occurred in only 21 per cent and it was marked or 'adequate' in only 9 per cent. In nearly half of the cases no imagery whatsoever occurred, and in presumably only one-tenth of the cases were the images 'fair representations of the actual sensations of movement.' "

Pertinent to this discussion are the experiments which Woodworth made on himself, as a result of which he reaches the conclusion that consciousness does not play an important part in the establishment of complete voluntary control. Woodworth was unable to detect any kinesthetic imagery in attempting to establish complete voluntary control of the isolated movement of extension and flexion of the great toe separate from that of the other toes. Quoting again from the author, he says: "I infer from the results of Bair combined with my own, that even in first getting control over a particular movement, at least in the case of adults, the kinesthetic image of that movement is neither a necessary nor a sufficient condition." The author calls attention to the fact that the child is equipped by nature with a large store of "definite reactions, defensive, locomotory, nutritional, vocal, ocular, facial, as well as with what seem more random movements of the arms and legs. These reactions necessarily occur first without the child's foreknowledge or intention; but having thus occurred they enter into the developing system of his thoughts and desires; they become associated with other things, and this is the process by which the child acquires control of his motor inheritance. The question is whether the first associations are formed between the movements and the feelings and images of them, or whether the child's first desire for a movement is the desire to feel it again." That this is not necessarily so is evidenced by a number of observations,

one of which is reported by Kirkpatrick.¹⁵ A child who was 17 months old and had not learned to walk, one day became interested in a pair of cuffs which had been left by her father on a table. Creeping to the table the child pulled herself up, reached for a cuff with one hand and put it on her other wrist, thus standing alone for the first time. Now she placed the other cuff on her other hand and walked across the room. When the cuffs were taken from her, she refused to walk any longer and it was only after a pair were given her that she resumed the new-found act of walking, which she continued to use even after the cuffs were taken away from her a few days later.

Instances of children who have learned to walk suddenly are numerous; for instance, my own first boy did not walk although he was 15 months old. Suddenly one day he straightened up from the floor and walked unaided for fully 16 feet. His aunt screamed with surprise and pleasure, and the boy immediately sat down and did not walk again until two weeks later. On this occasion some noise in the street attracted him and he lifted himself up by the window sill, stood there looking into the street and then turned around and walked across two rooms to his mother. These cases certainly show that kinesthetic images are not always the first thing to be associated with movements, and that they do not necessarily precede them. These acquired skilled acts, however, having once been mastered, the interest passes on to others. There probably is not much doubt that the initial acquiring of movements becomes the property of the child accidentally.

Dexter⁷ observed the progress made by a child in the coordination of movements. He noticed that this child one day happened to put his hand into his mouth, seemed pleased and attempted to do it again. The following day he succeeded a number of times and in a few weeks had the movement completely under control. Woodworth, in detailing this case, feels that it is impossible to conceive that the movement of the child's arm, when it had accidentally put its hand into its mouth, was accompanied by any particularly different sensations or impressions from other move-

ments which it had attempted prior to this of a similar character. Later, the sensations in the mouth having given pleasure, they became associated with the motor innervation and thus the movement finally became a voluntary one. This may be the genesis of a number of voluntary movements. The same argument is applied to the matter of visual and auditory images, the author insisting that the eye movements illustrated by stimulation of the visual cortex and the movements of the head in hearing due to stimulation of the auditory cortex may occur quite independently of the motor area. For instance, it is a fact that if the coordinating center for eye movements is stimulated, even if the motor area for the eyes has been cut off, the eyes will turn in the direction of visual stimulation. This would seem to preclude the necessity of these movements depending on kinesthetic images. This line of reasoning also applies to the turning of the eyes towards a source of sound, this movement also occurring on stimulation of the auditory area when the motor and visual areas are destroyed. Important for the production of voluntary movements, however, according to Woodworth, is the sensation of the initial position of a member, since the movement will differ according to the initial position. If a hand is in the position of flexion, that position must be sensed in order to determine the initiation of movement in response to stimuli. If the knowledge of the position of the hand were lacking, the result would be indecision. Sherrington²⁸ has shown that by cutting the afferent fibers from the muscles in monkeys, indecision and ataxia of movement occur. There will be over-reaching or premature stopping. This special sense of the position of the limb prior to the initiation of movement may not come into consciousness and it may be in a large measure reflex, but in deliberately preparing to make a movement this sensation of position is absolutely essential. Woodworth's conclusions are, therefore, first, that a kinesthetic image does not occur as the adequate determinant of voluntary movement. Second, he denies the existence of any image at all as such a determinant, and third, he insists that the field of attention just prior to the movement is often inadequate as a "disting-

guishing mark of the movement." He further states that the "intention" is not always present and is seldom felt in the field of attention at the moment just preceding the innervation of the movement. According to this author, then, the proprioceptive sensation of position of a limb is vital to the occurrence of voluntary movement. In passing, attention is called to the fact that the cerebellum is the great proprioceptive organ. Referring to his experiments, Woodworth found that in a great majority of cases the condition of mind preceding the movement was almost blank. He says that the "complete determinant of a voluntary motor act, that which specifies exactly what act it will be, is nothing less than the total set of the nervous system at the moment. The set is determined partly by factors of long standing, instincts and habits, partly by the sensations of the moment, partly by recent perceptions of the situation and by other thoughts lately present in consciousness. At the moment, however, these factors, though they contribute essentially to the set of the nervous system, are for the most part present in consciousness as a background or fringe, if at all, while the attention is occupied by the thought of some change to be affected in the situation."

All this is quoted in refutation of the theory set forth by Wilson in explaining apraxia.

On page 192 of the 31st Volume of *Brain* appears the following exhibition by this author of the theory of the production of apraxia which is taken from Kleist.¹⁶ The instance, as quoted by Wilson, is that of the sight of a rose, "suggesting the idea of my taking it and putting it in my buttonhole." In this scheme the diagram of Wernicke is used. "The *s* stands for a visual impression, the *A* would stand for the intimately associated idea, namely, the rose. The *Z* is the ideational connection suggested by the idea of the rose which is to put the rose in my buttonhole. Finally, *m* is the idea of the movement which is to work out the aim or object of the idea of plucking the rose." Now, the author says, follow the actual innervation of the motor sensory area and the completion of the act. In the light of Woodworth's experiments and the views which he lays down as a result of the same, one can see that this generally accepted scheme or diagram

explanatory of the production of purposeful movements, is untenable.

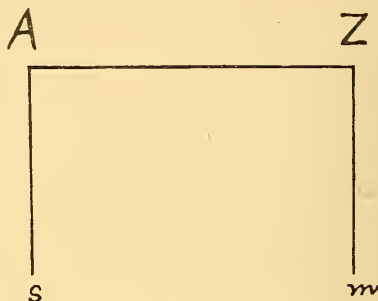


FIG. 3. DIAGRAM OF WERNICKE

CHAPTER V

We are now ready for what we believe should be the proper conception of speech. Having as we think proved that speech is a form of skilled voluntary movement and that it is a function of the entire intellect, it follows that the development of what we know as general intelligence or, psychologically, the mind, is inseparable from the development of speech and skilled voluntary movement. Speaking clinically, therefore, disorders of general intelligence, aphasia and apraxia are inseparable and indivisible. In order to prove this clinically, we must show that all cases of aphasia and apraxia are at the same time deficient in intellect. We must prove that will, memory, judgment, emotions, perceptions, impressions, everything that we include as a subdivision of the concept "mind," are affected in cases of aphasia and apraxia. We do not anticipate a great deal of difficulty in doing this. Not only must we show that intelligence is defective in cases of aphasia but we must point out this defect in all clinical entities wherein speech is affected; otherwise, our conception breaks down entirely. We have already illustrated this point in our cases of aphasia; later we shall bring to our aid cases of Friedreich's ataxia and Marie's heredo-cerebellar ataxia, cases of multiple sclerosis with speech defect, chorea, paralysis agitans, and the other clinical conditions in which speech is affected, not leav-

ing out the so-called functional conditions. In keeping with our conception we are prepared to show, clinically at least, that the entire brain takes part in the control of the mechanism of speech, not excluding the cerebellum, for in a number of these clinical conditions, this great proprioceptive organ is involved, if not primarily, at least secondarily. For instance, in chorea, Hughlings-Jackson, as far back as 1864 in the clinical reports of the Lincoln Hospital, pointed to the case of a patient suffering from chorea who for several years had been in the habit of saying quite involuntarily, "bloody." In recounting this instance, Jackson states that the patient was frightened by a man shouting the word. The fright "produced chorea, and, if I may use the term, chorea of his mind too; as for three days he said the word 'bloody' and little else, and now he ejaculates it occasionally. The mental process for saying that word is as little under his control as a few of the muscles of his face are, for the twitching of which he is now attending at the Lincoln Hospital." While we are on the subject of chorea, it might be well to state that Poynton and Holmes²⁶ found vascular and nerve cell changes in every region of the brain and brain stem in this condition.

It need hardly be mentioned that it is not difficult to point out the coincident development of dementia, speech defects and apraxia in Huntington's chorea. Even in simple Sydenham's chorea the great irritability, mental instability and sensitiveness which occur, at times amounting almost to a psychosis, are often associated with speech defects. These children speak in a quiet, tired, monotonous, colorless manner, or in jerky, sharp, explosive, syllabic stammering, particularly if the choreic movements are severe. According to many authors, choreic children are nervous, irritable and excitable, even before the movements make themselves manifest, and some show marked grades of feeble-mindedness. In the cases which afterwards develop Huntington's chorea, these characteristics are even more marked.

With this digression we will proceed to our discussion of the presence of mental defects in aphasia and apraxia. In the discussion of apraxia in relation to various forms of dementia, Pick²⁵ states that dementia is a mosaic of

partial dementias localized or localizable, and that this has an obviously important bearing on the causation of apraxia. We have seen, too, that Bonhoeffer¹ in his work with cases of alcoholic delirium in connection with apraxia found that acute exacerbations in alcoholic delirium may produce clinical symptoms referable to various areas of the cortex, and that he is thus able to correlate clinical symptoms in alcoholism with lesions in various parts of the cortex, as confirmed by autopsy findings. In all forms of the psychoses, particularly general paresis and dementia precox, aphasia and apraxia may occur. A number of cases of apraxia studied by the German workers in this field were in the presenile, sclerotic and senile dementia types. Most of Pick's²⁵ cases were arteriosclerotic and senile dementias. Other workers have pointed to the effects which the general destructive processes in dementia precox have on speech and voluntary movements of various kinds. Southard²⁹ showed in his work entitled "Study of the dementia precox group in the light of certain cases showing anomalies or sclerosis in upper brain regions," that the symptomatology in the cases studied might be explained by reference to the cortical localization of the most important disease processes found. In the pre-Rolandic group, this author places nine cases with injury more or less severe to the whole of both upper frontal convolutions. The symptomatology of this group is embraced in the terminology catatonia and paranoid dementia precox. The pathological condition found is gliosis and satellitosis; in one case (No. 1358), the symptoms were exclusively catatonic. In this case the greater part of both frontal regions was wholly spared from microscopic lesion. The only lesion found was one in the left inferior frontal gyrus. In the other eight cases, the entire brain underwent a mild generalized sclerotic process characterized microscopically by widely distributed glioses affecting principally the frontal and occipital lobes and including the cerebellar cortex and even the pons. In case No. 1062, the patient showed various disturbances of speech. He was abusive, delusional, noisy, obscene, and also had a prolonged period of mutism. In all of the other cases the clinical descriptions

are meagre, but frequent reference is made to the presence of catatonic symptoms or paranoid delusions with various speech disturbances. Therefore, speech, it can be inferred, was found correspondingly affected with the intellect in each case of this group.

In his post-Rolandic group, there were seven cases, four of which were alike in developing catatonic symptoms. In one case, No. 1137, with a sensory misinterpretation of stimuli with catatonia, the pathological findings were in the right supraparietal region and Southard states that "microgyria and the dense local disposition of fibrous tissue form a striking local lesion in this case." There were also, however, general gliosis and satellitosis. Another case, No. 1298, with catatonia and depression, which had at one time been diagnosed as manic depressive insanity, disclosed on autopsy a double postcentral gliosis in the white matter and visible atrophy of the right postcentral gyrus, also a peculiar focus of "durapial adhesions over the left middle frontal gyrus." Taking the whole of this group of cases, Southard says that they "show lesions which lead to the suspicion, if not the proof, of congenital anomalous symptomatology. These cases have catatonic symptoms in common." In an occipital subgroup of this post-Rolandic group, the author places three cases in which the occipital regions were more markedly sclerotic than the rest of the brain, and he points to the well defined "constantly recurring, voluntarily executed, visual episodes (communications with God) as a clinical anatomical correlation." However, in all these cases there were changes also in the frontal lobes. In one case of this group, No. 1294, the hallucinations shown by the patient were auditory rather than visual, and the brain showed frontal, cerebellar, and pontine glioses in addition to the occipital. Interesting to note is the fact that no temporal gliosis was made out macroscopically, but there were foci of leptomeningitis, both of the temporal and frontal types. In another case, No. 1317, although there was occipital microgyria, the hallucinations were auditory. There were some religious delusions. This would seem to break down the paranoid correlation which may possibly be made out in some of the other cases.

In an infrasyllvian group, Southard places

one case, No. 1319, with paranoid delusions. He was not able to show any anatomico-clinical correlations in this case.

In the cerebellar group, besides the cerebellar changes, there were profound and widespread changes throughout the whole brain in most of the cases. In one case, No. 1034, there was macroscopically pure sclerosis of the dentate nuclei of the cerebellum. Clinically, this patient had *flexibilitas cerea*. He began with paranoid symptoms and in six weeks became catatonic. He also had auditory hallucinations and persecutory delusions, was mute, reticent, impulsive, showed echopraxia and automatism. Although macroscopically there were no palpable changes excepting in the cerebellum, microscopical examination showed a widespread gliosis in various areas of the brain. There was also a satellitosis of the frontal region. In another case, No. 1168, besides the postcentral hypoplasia on both sides in their upper two-thirds, there was a sclerosis of the olives and of the right dentate nucleus and also of other portions of the cerebellum. Clinically, there were hebephrenia, mutism, resistiveness, impulsive reacting movements and attitudes. The largest group of cases reported by Southard was a group of fifteen which were clearly dementia precox, but macroscopically no lesions were found. Microscopically, these cases showed satellitosis of the deep cortex layers and a gliosis of the dentate nuclei. The author claims that a number of these cases might with propriety be classed with the cerebellar group.

In a summary of this material, Southard states that 68 per cent of a series of dementia precox cases, from which there were carefully excluded frank arteriosclerotic and coarsely wasting processes, showed macroscopical atrophies and apalias. In 86 per cent of the entire series, or in 24 out of the 28 cases studied, either coarse or microscopic lesions were found. Quoting the conception of Wernicke that insanity is a disease of the association limits of the brain, Southard says, "Even in the field of aphasia, one observes with recent writers a tendency to replace the focal destruction theory with some theory more consonant with modern ideas as to the tremendous breadth and complexity of the cortical areas attending speech."

In explaining convulsive movements, cata-

tonic attitudes, etc., the author says: "Neither a convulsion nor a catatonic impulsive movement is adequately explained by lesions of the muscles involved, by lesions of the motor nerves or spinal cord, by activities in the central gyrus, or even necessarily by any post-Rolandic or pre-Rolandic brain mechanism or combination of mechanisms. Perhaps, indeed, the reason lodges back of the central nervous system." For our purposes, it is well to quote the author's 12th and 15th conclusions:

"Several groups of cases were classified from the distribution of macroscopic lesions, although the focal purity of these cases can often be brought in question from the results of microscopic examination (infrastellate gliosis and satellitosis also in macroscopically 'normal' areas).

"I. Pre-Rolandic group, including a superior frontal-prefrontal subgroup of paranoid trend.

"II. Post-Rolandic group, including (a) post-central-superior-parietal (sensory-preceptual) subgroup in which katatonic features are the common factors; (b) occipital subgroup.

"III. Infrasyllvian group (too small for clinical correlations).

"IV. Cerebellar group (katatonic features).

"The cerebellar-katatonic correlation is doubtless in line with some contentions of the Wernicke school, and obvious comments might be made in connection with the proprioceptive functions of the cerebellum (Sherrington)."

A résumé of the deductions which we can draw from this study of Southard's would again seem to bring us confirmation of the fact that speech functions cannot be separated in any of their elements from the development of the mind or intelligence as a whole. It would appear that the author himself has not been able to show conclusively that the apraxia and aphasia and other speech defects seen in dementia precox are due necessarily to lesions in any particular focal area in either hemisphere. For the first time we have an indication of the part which the cerebellum may play as the chief proprioceptive organ for the coordination of voluntary movements, including speech. In all the catatonics of Southard's series, there was some degree of mutism, stereotypy, echopraxia or negativism of speech

and movement. In a number of his cases, the cerebellar cortex and its dentate nuclei were seriously affected.

Southard, in conjunction with Caravan,³⁰ presented two cases of chronic lesion of the left angular gyrus which received the clinical diagnosis of dementia precox. One was a cyst of softening and the other a solitary tubercle. It is well to remember, however, that these authors did not report microscopical examinations of any other portion of the hemispheres, brain stem or cerebellum. Both the lesions found were in the left hemisphere. According to the summary, both patients at one time or another showed hallucinations of hearing and seeing, disorder of consciousness, confusion, incoherence of speech, mutism, impulsivity, fixed attitudes, mannerisms, violence and catatonic stupor. A sample of Case A's speech is "Sure I am somebody. I must be somebody. Can't you see there is no bed? As there is no bed there is no room." Case B showed grotesque shifting of ideas with some play on words. "There is your knuckle, bite it and its pinocle. Look at the Spanish-fly blister." Together with the speech disorders of these cases, there were also emotional changes. These were volitional defects, and it is particularly stated by the writers that the patients showed little or no intellectual disorder. The conclusion is drawn that these two cases with findings in the left angular gyrus tend to correlate the clinical symptom complex of dementia precox reactions with catatonias with lesions in the left angular gyrus. This conception of these cases with no microscopical report available for study of other parts of the brain other than the immediate focal lesion found, is unfortunate, particularly in view of the further fact that Southard's larger series of cases do not by any means seem to warrant the correlation of clinical symptoms of catatonic mutism, etc., with focal lesions anywhere in the brain. It would appear plainly evident from a consideration of this autopsy material that the apraxias and aphasias and other speech disturbances occurring in dementia precox are not susceptible of correlation with pathological findings in any so-called centers for speech or for voluntary movement. It is noteworthy that Kraepelin¹⁷

separates a large group of his dementia precox cases with marked apraxic and aphasic symptoms and calls this group "schizophasias."

Charles I. Lambert¹⁸ reported some clinical and anatomical studies in Alzheimer's disease and related conditions. Five cases were reported, one of which may have been a case of Korsakoff's syndrome. In all five of them, the author states that "among the more striking symptoms of these cases were the outstanding symptoms of agnosia, aphasia and apraxia. An exact analysis of these focal symptoms presented considerable difficulty, since to an impaired recognition, word understanding and expression, there were added a general dementia and an unresponsive resistive attitude." The author further adds, however, that there was "a complete loss of spontaneous speech without gross paralytic symptoms developing, so that we might probably conceive of the aphasic symptoms resting largely on the marked dementia present and somewhat in the nature of a mixed sensory-motor-transcortical aphasia. When apraxia symptoms occurred they were mainly of an ideatory nature. In relation to the aphasia and apraxia disorder, the absence of paralysis or even limited impairment of motor function is of interest and should be remarked in relation to a differential diagnosis." In one case there was a right hemianopsia. It does not seem necessary, in view of these statements by the author, to give illustrations of the aphasic or apraxic status of these cases; but a close study of the clinical findings leaves no doubt that both these conditions were present.

The anatomical findings in these cases are again brought to bear as an aid to our contention that intelligence in its widest sense, apraxia and aphasia are one and the same process, that they develop together and disintegrate together. Quoting from the report of the autopsy findings, particular note is made of the fact that generally distributed or circumscribed brain atrophy is present with the complete absence of arteriosclerotic change in the vessels to account for it. "The central convolutions in all cases appeared almost normally full, the frontal convolutions atrophic, the temporal, parietal and occipital convolutions rather strikingly atrophic in two cases, par-

ticularly so in three of them." No areas or foci of softening or gliosis were seen or felt. Sections of the vessels shown by the author present no changes in any of the cerebral arteries or their branches of an arteriosclerotic nature. There were changes in the muscularis and in the perivascular lymph spaces. Microscopically, gitter and granular cells were found. No mesodermal cells were found in these spaces or in the pial spaces in the cortex. In the various layers there were changes obscuring the normal layer formation, reduction in the number of cells and an increase in the neuroglia elements, closely corresponding to the degree of atrophy present in the macroscopical examination. The fewest changes were found in the central convolutions; the largest degree of change was found in frontal, temporal and occipital lobes and in the cerebellar cortex. In the nuclei of the cerebellum the changes were also abundantly evident. Plaques of degeneration were also found in the spinal cord, in the brain stem and in the basal ganglia. The plaques were found evenly and symmetrically distributed throughout the two hemispheres in two cases, and asymmetrically distributed in two other cases. In one case (the author's case No. IV) it is said that the left parietal lobe in the angular gyrus shows a maximum degree of macroscopical and microscopical change. In this case the first layer of the cortex in the area mentioned showed changes which were quite striking. It is of interest to note that in this case the first evidences of disease appeared in the sixtieth year "with a change in his personality and declining memory. He failed to recognize old friends, was vacillating in his mood, sometimes agreeable, again irritable and obstinate. He ate ravenously and wet and soiled his clothing." In talking, the patient repeated many words and at times was unable to finish what he was saying. His talk was a jargon or gibberish. This case had a right hemianopsia but no paralyses. Further examination shows that this patient "paid no attention to simple commands. He shook hands when the physician held out his hand, put out his tongue when told to do so, tried to open a knife, and when given a clock said '20-100-100-20'." This patient was aphasic and paraphasic, but not distinctly apraxic. That he had agnosia, how-

ever, is shown by the fact that one day he put his hand into the hot stove without appearing to know what he was doing. He did not know what food was for, had difficulty in carrying it to his mouth and had to be spoon fed. The inference is that he tried, however, to carry the food to his mouth. He is said to have had difficulty in handling a pencil when asked to write, but no mention is made of the fact as to whether he could write before his trouble. On occasions he put on a sheet as a shirt and would pull and haul at the bedding by the hour. The autopsy in this case showed more particularly a lobal atrophy involving the left parieto-occipital lobe, although there were brain changes in practically all parts of the brain. One can notice even with this author, who seems to have made a very careful anatomical study of these cases, the tendency to correlate a clinical finding with what he considers the most important pathological lesion without due regard to the conditions in the rest of the brain.

Without going any further into the pathological and anatomical studies of these cases, we have, therefore, five patients suffering from Alzheimer's disease characterized chiefly by aphasia, apraxia and agnosia, which, instead of confirming the presence of focal and diagrammatic cortical localization areas for speech and voluntary movement, rather seem to confirm our contention, which we wish to repeat, namely, that the function of speech and voluntary movement is an integral part of the development of the functions of the entire brain, and is incapable of being anatomically, physiologically or psychologically separated into centers.

CHAPTER VI

The disturbances in speech seen in the various functional conditions, such as the neuroses, the hysterias, fatigue and the emotions, are of great interest in linking up this function with that of the general intelligence of the individual. Trigant Burrow⁴ has attempted to correlate the development of hysterical manifestations in a phylogenetic way with the development of speech. He develops the conception that the origin of language was mimetic and that the

language of primitive races was essentially instinctive and gesticulatory. The connection between inner thought and outer action is inherent and inseparable, and language, as it developed, was used to disguise whatever was unpleasant. He also draws an analogy from this for the mechanism of the production of hysteria.

In hysteria, the symptoms develop as a disguise of unpleasant conflicts, desires and strivings. At any rate, it is certainly true that very few cases of profound hysteria exist without some sort of speech defect. Whether we believe with Freud and his followers that hysteria is a conversion mechanism by which special subconscious conflicts are converted and enervated into somatic symptoms, or whether we believe that the symptomatology of this condition is due essentially to a psychical dissociation and an oversuggestibility to inner or outer influences, is, for the purposes of this discussion, not important, nor is it important to believe with Burrow that hysteria is a phylogenetic reaction, or with others that it is essentially a mechanism of reaction to fear and other unpleasant emotions. Whatever the mechanism of the production of this condition, it is nevertheless true that all sorts of speech manifestations occur which vary from complete aphonia or anarthria to stammering, explosive or halting speech or even slight dysphonias and inconstant aphonias. It is also important to note that hysteria occurs in individuals with unstable emotional control possessing also defects in the volitional and inhibitory spheres. Two cases illustrative of speech troubles seen in hysteria are given as follows:

The first was a man 40 years of age, single, a carpet layer by occupation, who on June 2, 1917, entered a carpet beater to extract some carpets. This machine is a large steel cage which is whirled around by electricity, so that the carpets contained therein may be mechanically agitated and thus freed of the contained dust. In some manner he was locked in the cage, the machine was started and he was unable to get out. He was whirled around inside of this cage and knocked from side to side, for about half an hour, the carpets saving him from being mashed into a pulp. All the time he was in the cage he yelled and screamed in terror for

help. Finally he was released, and bleeding from a scalp wound on his head staggered from the cage supported by some fellow workmen. He trembled and quivered with fright, stuttering and stammering fearfully. Suddenly, breaking from his rescuers, he ran out into the street; he was caught and brought back with great difficulty. He was finally calmed and taken to a hospital where his scalp wound was stitched; he did not speak at all for three hours after the accident. Following this, he returned home trembling, fearful and very much upset. That night he lost his voice entirely and did not speak for several days. The neurological examination disclosed no palsies anywhere and absolutely no evidence of paralysis in the distribution of the 9th, 10th and 12th nerves, so there was no physical reason why he could not speak. There was no evidence of a motor or sensory aphasia, no paralysis, and absolutely nothing organic to explain his condition. At the end of the third day when he began to speak he talked in a harsh whisper. He suffered from generalized tremor. Eventually, he was able to speak more strongly, but he has continued to this day with a defective speech characterized by harshness, quick changes in tone, pitch and cadence, halting, explosiveness and tremulousness. He talks like one who is in the throes of a terrible fear, and speech is associated with an increase in the generalized irregular tremor throughout the body.

Another case is that of a young man of 20, who fell from a scaffolding while painting, a distance of about 25 feet, and sustained what was said to be a concussion of the brain. He was not unconscious following the injury, but was stunned and dizzy. He went home unassisted and several days later became affected by an impairment in his speech. There were syllable stuttering and tonic and clonic stammering. At times, however, when the patient's confidence was gained and he became greatly interested in a conversation, he would speak several sentences without any trace of stuttering whatsoever. On several occasions he made attempts to return to his work. Not only did his speech defect become worse, however, but he trembled and became so nervous and excited that he had to give up working. Treat-

ment in both of these cases was of very little benefit, and neither one of them has improved very much.

Everyone who has been fatigued will notice how slow all his mental processes become. Associations are awakened slowly, recollections are delayed, and active thought production is difficult. So also the expression of thought will suffer, the speech becomes slow, colorless, lacking in emotional tone, and there is a slurring and sliding in the speech with paraphasic inability to choose the proper words and phrases. In this connection, it is pertinent to refer to the five cases of pseudoparesis developing in young soldiers reported by J. Ramsay Hunt at the November, 1916, meeting of the New York Neurological Society. Hunt described five young individuals who broke down under the intensive training, and the physical and mental stress incident thereto, in a cantonment where he had them under careful observation. The symptomatology suggested paresis. There was tremor in the muscles of the face and tongue, sluggish reactions to light in both pupils, elision and slurring speech and syllable stuttering, marked memory and attention defects and a general slowing of the mental faculties of rather rapid development. The serology was negative, and he was able to exclude syphilis, to the satisfaction of himself and everyone who heard him. All these five cases recovered under rest and proper treatment, the defects in speech and intelligence cleared up hand in hand, and all of them returned to their duties perfectly well at the end of a month.

Hoch¹¹ calls attention to what he terms the lowering of mental tension in fatigue and the aphasic and paraphasic characteristics of fatigue thought processes and speech. An amusing incident is brought to my mind of the suspicious and alarmed actions which an intern on my service in one of the large hospitals in this city showed when he heard me speaking about a case which we were discussing at the bedside. I had been under a very prolonged physical and mental strain and was very much overworked. The journey to the hospital had been very tiresome, and I was extremely fatigued. I had anticipated an easy hour of round-making, but case after case of interest

had been called to my attention, and we finally arrived in the ward where a patient lay in whom the intern was very much interested. He asked me to make the examination with him and help him in the diagnosis. The patient, who was a paretic, presented a very beautiful illustration of perseveration of speech and echolalia. In using the test words to bring out also the typical paretic speech defect, the intern was very much horrified to see that I butchered the test words in a much more striking manner than did the patient himself. Everyone who has been fatigued has had an experience of this kind. Public speakers with a reputation for clear diction and beautiful enunciation will often notice the difficulty they have, when fatigued, not only in thinking but also in speaking. Famous public orators often complain that the last speech which they make at the end of a hard night of campaigning is always the most unconvincing, dull and poorly enunciated. One such speaker has described very vividly to me his feelings at such times. He recounts how he starts out after dinner at his first meeting full of vim, the thoughts following one another in quick association, with logical sequence and telling effect on his audience. Toward the end of the evening, at some times earlier than at others, as he gets tired, although he repeats with very little variation the same speech which he has delivered earlier in the evening, the thoughts expressed are not so logically and clearly put and do not follow each other with the facility by which his earlier talks are characterized. He has on a number of occasions become panic-stricken when confronted with the difficulty of pronouncing words which he ordinarily speaks with great distinctness and clearness. Words are slurred and mispronounced and he finds it impossible to give them the proper emotional emphasis. Often on such occasions he has to cut short his discussion and give up for the evening.

The emotions also play an important rôle in the development and expression of speech. The inhibitions and accelerations of speech and thought production which occur under emotional stress are known to all of us; even on the motor side of speech the element of emotional tone plays an important part. In-

deed, it is almost impossible to separate this element from intellectual speech. It is inconceivable that any motor formula alone can be responsible for speech when one considers only this single influence of the emotions in speech. The simple words "No" and "Yes" can be delivered so that a variety of meanings may be implied and understood. Skilled actors are able to throw into the mechanical production of speech feelings of all shades, and one may often be thrilled to passionate heights merely by listening to the clever play of emotional tone which such artists are wont to use in their delivery. The emotions, therefore, together with the rest of what goes to make up the mind, are inseparably connected with speech, not only on the intellectual side but also in the motor expression of that function. Hughlings-Jackson¹² seemed quite satisfied that the right hemisphere controls the production of emotional and automatic speech. He and others have pointed to the fact that even with extensive destruction of various parts of the left hemisphere, aphasic and some apraxic individuals were able to perform involuntary movements or make involuntary utterances which could not be repeated voluntarily. Such individuals will use an explosive "No" or "Yes" in assent and dissent, or will swear volubly when confused or angered but will not be able to repeat the swear words when asked to, even from dictation. One of my patients, J. M., used in Chapter III, to illustrate the presence of intelligence defects, when exasperated one day, swore a blue streak in perfect English but was never able to repeat the swear words.

In this connection, it is well to call attention to the emotional lack of control of certain aphasics. There are two patients now in the wards of the Kings County Hospital who show this very beautifully. One is an old man with a diplegia who is prone to prolonged attacks of involuntary laughter or crying, leading almost to syncope from exhaustion. The proper emotional responses are seldom or never given correctly. For instance, one day when he was quite sick his wife came to console him. She was very much touched and cried as she spoke comforting words to him. All during her visit he laughed very forcibly and uncon-

trollably, his face becoming suffused and his breath coming in gasps, until complete exhaustion caused his involuntary laughter to cease. On another occasion, as a test, I told him some supposedly very amusing stories. He immediately began to cry, the tears rolling down his cheeks, and did not stop for a half hour or more. When questioned, he explained very carefully that he did not desire to cry when he cried nor to laugh when he laughed, that he really felt like doing neither but was unable to control his emotional responses. Sometimes when he simply desired to smile, the smile soon developed into a loud, gradually increasing guffaw and finally into convulsive laughter. This patient was almost wordless. He could say only "Yes" and "No," using both these words properly for assent and dissent. He could read print and could write from dictation and from copy, and could explain in writing very well what he had read. He could write with his (left) paralytic hand and showed a very beautiful mirror writing. He saw and understood what he saw. There was no hemianopsia. He heard and understood what he heard. He recognized the names and uses of objects, and could do simple sums of addition spontaneously. The difficulty seemed to be chiefly on the motor and emotional side of speech. The patient was right-handed.

Another patient, in the same ward, was a Portuguese sailor with a left hemiplegia. This patient never cried regardless of what was said to him. He was always ready for a smile which soon grew into a fit of uncontrollable laughter. The history in this case is not reliable because he was brought to the hospital wordless and paralyzed and no relatives have ever been found. We do not know whether he was right-handed or left-handed. He speaks a senseless jargon spontaneously. He uses properly several simple phrases. For instance, he will say in French or in Spanish, "How do you feel?" "I feel good," "Yes," "No," "I feel fine," "I thank you." In English he can say, "I am all right," "Yes," "No," and "My name is Emanuel; I am a sailor man." This is as far as his spontaneous speech production will allow him to go. There is no hemianopsia, but there seem to be visual and auditory agnosia and paraphasia. He is unable to recognize the

names of various objects seen and heard. He cannot write, and attempts at getting him to read are met merely with guffaws of laughter. He performs simple imitative movements, can light a cigar properly, will wind a watch on command, will extract a match from a box. There is no tactile agnosia. This patient has never been known to cry during his entire residence in the ward. He will laugh even if things are stolen from him or if he is berated and scolded. These two cases show quite clearly the intimate relations between the emotions and speech.

In conditions of great emotional stress, speechlessness is very often seen; fright, anger and great joy will very often impede speech, particularly in women. These emotions may have a contrary effect on speech; but whatever the individual reaction is, speech is always somewhat affected in conditions of stress. Some persons who are habitually accustomed to speak quickly, under the influence of rage will talk quietly, slowly and incisively. Others will speak rapidly, ungrammatically, stumblingly, with stuttering or stammering of various types.

CHAPTER VII

That auditory word images, visual word images or kinesthetic word images are not essential in producing speech, can be seen, aside from the psychological considerations mentioned, from the following points. People who have the faculty of expressing themselves by means of changes in tone and modulations of voice developed to a high degree, find it possible to transmit meanings by such manipulation of spoken words which the utterances themselves may belie. For instance, talented actors, by voluntary control of speech, are again and again able to impart to the simplest phrases importance which others cannot do. This, of course, does not altogether depend on the quality of the receptive auditory apparatus of the hearers. Whether one catches the meaning imparted to these words, other than the words themselves, depends almost entirely on the attention and degree of insight which the hearer possesses. As an example, it is possible for a talented individual, and for many others,

uttering the words "I hate you," even in the dark, where associated gestures cannot be called upon to aid in giving a contrary meaning to this phrase, so to say these simple words as to convey to the hearer exactly the opposite state of feeling on the part of the speaker. It can be seen, therefore, that the hearer must be exercising in the fullest possible way all the functions of the intellect in this particular case, especially the ability to interpret the emotional elements of speech, in order to obtain the intended meaning of the speaker.

Gestures, of course, are a great aid in speech. Calling again on examples seen in everyday life for illustration, all of us will readily recall occasions when a shake of the head or movements of the eyes, of the face, or a general attitude of the body during a conversation, meant more than the actual words spoken. Indeed, it is possible to convey a great many thoughts in this form of speech without the actual utterance of a single word. Shall we exclude, therefore, this form of thought communication and expression from the category of speech? It seems, rather, more logical to incorporate this method of communication between humans as an integral part of the speech mechanism.

In analyzing the processes through which the individual conveys thought by means of gestures or changes in tone and cadence of spoken words, one cannot say that kinesthetic memories, visual memories or auditory memories of words are being used to help either the user of these forms of expression or the hearer in the understanding of them. It is impossible to analyze separately these various images in speech because they are not, according to our view, analyzable into separate components. They are all part of the general functions of the mind.

It has often occurred to us to ask of the psychologists who would have us believe that various sorts of images are necessary or essential in diagrammatic operation for the proper perception or expression of thought, how they explain the understanding of difficult abstract or scientific propositions as laid down, for instance, by teachers in giving instruction to their pupils. A professor, in expounding the theories of the physiological principles involved

in the action of the glands of internal secretion to a group of medical students who have never heard anything about them before and have never seen them, is able to give an accurate exposition of his own thought so as to make clear to them the knowledge which he wishes to impart. It is impossible to say beforehand whether such students are by preference so-called visualists or auditifs. The impressions received by the students and the amount of understanding of the matter which they have of such impressions received, depend entirely on the degree and quality of their general education or general individual intelligence, and not on the calling up of any specific images. All the faculties of the mind must be used in receiving these propositions as they are being laid down by the teacher. It is inconceivable, as some would have us believe, that certain individuals arouse auditory word memories of what is spoken to aid them in the understanding of speech, or that others arouse by preference visual images of these matters. It would seem more in keeping with the probable truth that all possible sources of mental activity for the reception and storing up of impressions are used in such a case. If this were not so, the understanding of thought as it is expressed in speech could be made a simple matter, reducible to a measured capacity for imparting knowledge and for its reception, by means of the stimulation and production of various sensory images, whereas, as a matter of fact, we know that the ability to impart knowledge by means of speech and to acquire it by means of thought depends entirely on the individuals who are teaching or receiving this knowledge.

All these considerations would seem to point to the fact that general intelligence is the basis of speech and not any separate faculty of the mind. As a corollary to this, it must of necessity follow that no particular part of the brain and no group of centers can be said to control the mechanism of speech production in its broadest sense, although the control of the motor part of the speech function may very well be limited in a poorly circumscribed manner to a region adjacent to the motor area in the cortex for the muscles of phonation, voice, lips, etc.

CHAPTER VIII

As examples of speech disturbances due to injury, disease or maldevelopment in the afferent cerebellar tracts, we have the difficulties of speech seen in Friedreich's ataxia. As illustrations of defective speech seen in injury, maldevelopment or disease in the efferent cerebellar tracts, we have the heredo-cerebellar ataxias and the cerebellar types of catatonic dementia precox. Speech disturbances seen in the cerebro-cerebellar conditions are best illustrated by the cerebro-cerebellar diplegias and in some cases of multiple sclerosis. As evidence of speech troubles, using the word speech in the widest sense always, we also have the conditions which depend on interruptions in the tracts known as the extrapyramidal, namely, those connecting the corpus striatum and the red nucleus and, therefore, indirectly, the red nucleus and the cortex by means of rubro-cortical or cortico-rubral tracts, and the cerebellum by the superior peduncles. Illustrative of this are the speech disturbances seen in paralysis agitans and Wilson's disease.

Friedreich's Ataxia.—Here the disturbance seems to be, in the beginning, at any rate, one of loss of coördination explainable on the same grounds that the ataxia in the muscles of the trunk and extremities is explained; that is, that the afferent impulses to the cerebellum are cut off in the case of speech from the muscles of phonation and articulation. When these muscles are set in motion during utterance, ataxia occurs, resulting in the so-called "hot potato" speech. One of my patients described her difficulty by saying that she did not seem to be able to get her tongue and lips in the right position to speak properly, and at times she felt that it was impossible to speak at all because of this difficulty; but if she exercised a great deal of care and voluntary effort in speech and spoke slowly, she was able to speak better. The speech disturbances in this case consisted of a peculiar, slow, colorless, unemotional character in her speech, with changes in tone and pitch, occurring often in the same sentence, but nearly always in the course of speaking several sentences. It was quite customary for her to speak in a fairly even, slow, monotonous tone the first few words of the sentence, and

then to speak rapidly and in a different tone and to end finally in an entirely different pitch. There would be considerable volume to the speech at first, but this gradually tapered off as though the patient were tired. The most important thing seemed to be the entire lack of color in the speech, there being no emotional manipulation of speech possible whatsoever.

Another patient in the same ward had a speech which is described as halting, distinctly ataxic, with remarkable changes in pitch and tone occurring in the same sentence. Her voice also grew very weak towards the close of a long sentence, and words were spoken almost in a whisper at the end. Her speech was also colorless so far as emotional tone was concerned.

While both of these patients were advanced cases of Friedreich's ataxia, the peculiar character of the speech disturbance when it occurs early in this condition was seen in a boy of 12 years whose symptoms were only of three years' duration. The emotional tone in this boy was well preserved, and it was possible for him to show in his speech the quality of the particular mood in which he happened to be while talking; but throughout the speech there was an unsteadiness in holding sounds, more particularly vowels, as in Ah and A. These vowel sounds were pronounced in a very tremulous and unsteady sort of way, with changes of definite character in tone and pitch. It is interesting to note that in the first two cases, which were advanced, there was an associated secondary mental defect due quite possibly to the narrow institutional life which they led. In the boy, however, this was not observed; he was bright and did well at school. The first two, however, were distinctly retarded mentally in all intellectual spheres.

One other case of Friedreich's ataxia with speech disturbance is that of a young man of 26 whose symptoms began 18 years ago. He has the typical "hot potato" speech, talking as if something disagreeably hot was being held inside the mouth which had to be pushed from place to place during speech. Aside from this defective feature his speech is also slow, halting, with a tendency to unevenness in tone and is absolutely colorless emotionally.

Three other cases of this condition observed

by the writer showed no disturbances of speech whatsoever. They were very early cases, the symptoms being of not more than three years' duration.

Heredo-cerebellar Ataxia.—Three cases which I have personally observed of this condition occurred, one in a child of nine and the other two in brothers who were more than 40 years of age. There was also a sister who suffered from the condition, but she had no trouble with her speech mechanism. All three of these patients had the characteristic monotonous, halting, colorless type of speech, which is a great deal like the speech of the paralysis agitans cases. No matter how great the distress or the joy of these patients, they seem unable to convey an inkling of their emotional status through manipulation of their utterances. The words are pronounced slowly with a halt between each word or phrase, but there are no changes in pitch or tone. There is a tendency to slurring of sibilants and a strange lack of expression, not only in their voices but also in their faces. In this respect, I have been able to see very little difference between the speech disturbances of paralysis agitans and cerebellar ataxia. In the two brothers, there was a considerable degree of dementia of the organic type, defects being present in all mental functions.

Cerebro-cerebellar Conditions.—In cerebro-cerebellar diplegias, in dementia precox and also in multiple sclerosis, it has been definitely established that disease processes may occur in both the cerebrum and the cerebellum. In cerebro-cerebellar diplegias of profound type such as one sees in cases of accidental feeble-mindedness following difficult labor or other injuries or diseases soon after birth, there is very evident agenesis in both the cerebral and cerebellar cortex. It is rather common in these cases to have as an expression of the profound mental retardation an entire absence of speech. In milder types where the agenesis or defective development due to injury is not so profound, speech is retarded in direct proportion to the degree of brain atrophy or injury. The following case is illustrative of the former type and is reported in full from the records of the case taken by Dr. O. S. Strong, not only because it serves as a good

example of the condition mentioned, but also because it is of interest owing to the fact that speech has not developed at all even though the child hears perfectly.

A. A., male, five years old. Is one of twins. After a normal labor the child appeared to be all right. When fifteen days old, it began to have attacks during which its face would become blue and its body would be bent in a position of opisthotonus. These attacks occurred for two months; they were in the nature of generalized convulsive seizures and always began with a sharp cry. The child's body would be contracted tonically and then would bend backward, resting on the back of the skull and pelvis; the face would become very blue and the whole attack would last about ten minutes. Occasionally the attacks consisted merely of spasmodic crying with blueness of the face without convulsions. He had altogether three severe convulsions and many crying spells with blueness. Thereafter, the child appeared to develop fairly well, although he did things awkwardly and performed voluntary movements with a great deal of tremor and trembling. He did not creep until he was two years old and did not walk until he was three. Investigation shows that deliveries of both this boy and the twin were normal in every respect. The first teeth appeared when the child was about sixteen months old, three months after the normal child had his first teeth. The affected boy began to make sounds when he was four years old, but never distinctly, so that his vocal sounds were never understood as words. There has always been more or less slobbering, and the child has been irritable and hard to manage.

On the back of the child's head there are two large bony prominences, the one on the left side being much larger than the one on the right. The head is distinctly microcephalic and asymmetrical. The affected twin is in every way smaller and less well developed than the normal one.

The neurological examination shows that all the cranial nerves are normal. The fundi are normal. The pupillary reactions are physiological and hearing is normal to both air and bone conduction. The boy walks in a very

unsteady manner with the legs widely spread and with unsteady, jerky movements of his upper extremities and trunk. In stopping there is a tendency to fall to either side. He also stands very unsteadily when his feet are put together, even with his eyes open. All the superficial reflexes are normal; the muscle strength seems to be normal, although coöperation was so poor on account of his mental condition that nothing definite can be said about this. There is a tremor in both upper and lower extremities of the intention type; irregularly ataxic and choreiform movements are brought out very easily. There is distinct equilibratory and non-equilibratory coördination in both upper and lower extremities. There is marked adiadochokinesis. There are no sensory changes. Guttural sounds are made but no understandable word has ever been spoken. In other words, there is a very low grade of imbecility. The child is extremely emotional, bad-tempered, and not amenable to discipline, persuasion or correction.

The speech disturbances in dementia precox have already been mentioned. Reference will be made here only to the fact that Kraepelin¹⁷ has recognized a distinct form of dementia precox, which he calls "Schizophrenia," in which there are very marked changes in speech with aphasic and paraphasic characteristics. Kraepelin also dwells on the fact that speech disturbances may and often do occur in all types of dementia precox. The cerebellar findings in this condition by Southard and others should also be borne in mind.

Multiple Sclerosis.—In this condition the speech defect is probably a cerebellar disturbance. Indeed, in the paraplegic types of multiple sclerosis with the difficulty limited fairly definitely below the bulb, speech disturbances never occur. The opposite, however, is true where the sclerotic plaques are located either in the medulla or in the cerebrum and cerebellum. The speech disturbances range from absolute anarthria or aphonia to varying degrees of dysarthria and dysphonia. In these two types, the lesions are undoubtedly situated either in the cortico-spinal tracts and their afferent fibers to the cranial nerve nuclei, or, very rarely indeed, in the nuclei themselves; in the former case the speech is a spastic diffi-

culty. The typical speech of multiple sclerosis, however, is not of the bulbar type but has more of the cerebellar characteristics; namely, it is monotonous, peculiarly ataxic, colorless and emotionless, and shows frequent changes in tone and pitch.

F. W. Scripture²⁷ makes a study of the records of speech in disseminated sclerosis by the graphic method. In his conclusions on the nature of sclerotic speech, he notes the peculiar vibrations, irregular rhythm, monotony, etc., and attributes these qualities of the sclerotic speech to ataxia. When thickness and explosiveness occur in the speech, these symptoms are attributable to spasticity in the muscles of enunciation and are in no wise different from the speech defects seen in infantile cerebral diplegia or ordinary hemiplegia. In brief, Scripture attributes all the difficulties of speech in multiple sclerosis to ataxia or disturbance of tone or a disturbance of breathing, which might result in "staccato speech." He insists that scanning speech never occurs and explains that the disturbance of rhythm in the speech of multiple sclerosis which simulates scanning is explainable by an ataxia of breathing. Scripture distinctly states that syllables are not separated by pauses in this condition. This author feels that the laryngeal ataxia responsible for most of the speech disturbances in multiple sclerosis occurs also in Friedreich's disease and in some of the other conditions not yet thoroughly studied.

Paralysis Agitans.—In this condition there is characteristically found the same dull, monotonous, slow, but not halting speech, void of all emotional coloring, that is seen in cerebellar conditions without the true ataxia already spoken of in the speech disturbances of Friedreich's and multiple sclerosis. The speech is more apt to be stiff and is probably not wholly cerebellar and indeed may be in most part a spastic speech. Differentiating it from the speech of Friedreich's ataxia, is the fact that changes in tone never occur. There is a true monotony, with no halting or slurring, such as is seen in the heredo-cerebellar conditions.

In Wilson's disease, a report of a case of which is herewith appended, the speech is evidently gradually lost as a part of the gen-

eral mental deterioration with final dementia. The motor quality of the speech is undoubtedly spastic. This girl spoke through her teeth, very stiffly, and the only sounds uttered were throaty, guttural noises, only a word here and there being recognizable.

S. F., female, aged 22, single.

FAMILY HISTORY.—The mother, who is 45 years of age, is neurotic and suffers from headaches and metrorrhagia. The father died of tuberculosis (?) at the age of 30 and was a shiftless man who would not work. The mother of the patient finally deserted him and after his death married again. She had by Sadie's father three children, of whom the patient is one, the other two being a girl, 24 years of age, and a boy, 20 years of age, who are both said to be physically and mentally normal. The mother has had no children by the second marriage, and she states further that there is no history in any of the branches of her family of any illness such as exists in our patient. She knows very little about her first husband's family, and is not even sure that he died of tuberculosis, although she heard that he died from some wasting disease. The patient's mother states that the pregnancy as a result of which our patient was born was perfectly normal and uneventful and that the labor was easy and no untoward incidents occurred during the labor or the puerperium. The child walked at one year and progressed satisfactorily. She was bright at school and left when she was a little more than 12 because a cousin who brought her up thought it best that she go to work. She worked as a messenger girl in a dry goods store for several years and then stayed at home to keep house for her mother.

HISTORY OF THE ONSET.—The girl, having previously been perfectly well, when about 15 years old, suddenly fell to the floor and was carried unconscious to the bed bleeding profusely from the vagina or the rectum, her mother did not know which. It is said that the patient passed large clots of blood and that the hemorrhage lasted several hours. At the end of this time it stopped completely, but she lay in bed and was so extremely weak that she could not move her head and had to be fed.

Several days later when she finally began to talk and move a little, she complained of a severe pain in her throat and in the upper part of the chest. This pain was so severe that she was doubled up, her head and knees touching during the paroxysms. She still remained in bed, but gradually the pain subsided somewhat only to return at the end of several weeks with increased severity. She was taken to the Long Island College Hospital at the end of two or three months. Soon after this, she became drowsy and would lie in bed for months without moving and in a semi-conscious state. During this period, the pains in the throat and chest were not evident, but at the end of each drowsy period, they would return with increased violence, and during the attacks of pain, she would not be able to talk at all and was doubled up in a knee to chin posture, which was the only position in which she could obtain relief. During one of these attacks, she completely lost her voice and has never since regained it, not having spoken a word distinctly for more than four years. The dysarthria and dysphonia have remained stationary, there being no remissions whatsoever.

The tremor began three or four years ago. The grimace or spastic smile which the patient has, began about seven or eight months ago. Of late years, the patient has occasionally lost some blood from the rectum. On several occasions, her legs have become so extremely spastic that they have swelled considerably, although, as the patient's mother expressed it, "there was no dropsy and the skin did not pit."

The mental deterioration has been very rapid. The mother says that she is definitely sure that until seven or eight months ago the girl was mentally as keen as she ever was, despite the fact that she could not talk.

INSPECTION.—The patient is fairly well nourished, and lies in a spastic attitude, the face being fixed in a fast grimace or grin, the legs stiffly extended, the head and trunk also being held rigid. Her movements are made with stiffness and tenseness in the muscles, which are quite noticeable.

COORDINATION.—On account of the great

spasticity in the skeletal muscles, examination to bring out the coördinating status is not always satisfactory, but non-equilibratory tests, namely, the finger-finger, finger to nose, knee to heel and knee-heel tests, seem to be well done.

GAIT.—The patient can walk a little with considerable spasticity and is apt to fall, especially when she attempts to run.

SKILLED ACTS.—Are very poorly performed because of the spasticity, and speech is extremely difficult. She cannot write because of the tremor. There is aphonia and anarthria, although at times she can speak one or two words and will hum songs, so they can be understood.

REFLEXES.—All the reflexes are equal and active, both superficial and deep. There is no ankle clonus, although tapping of the patellar reflexes is sufficient to cause an extremely marked response with apparent clonus. The superficial reflexes, including the abdominals, are present and equal. It has been possible, on account of poor coöperation, to examine this patient's plantar reflexes only during sleep; and on a number of occasions when this was stealthily done at night, there was the normal flexion of the big toe and no Babinski was elicited. There was no Oppenheim and no Chaddock. The abdominals were present and equal during sleep. Pupillary reflexes are equal and prompt, both as regards light and accommodation, although there is a weakness in convergence, the left ocular movements not taking part in this reaction, the eyeball seeming to remain fixed with the same degree of spasticity as the skeletal muscles. The corneal reflex is equal and active. There is no nystagmus.

MUSCLE STRENGTH.—Does not seem to be impaired. The tendo achillis and the muscles of the wrist are held so rigidly as almost to constitute a deformity, and it requires great strength to overcome the spasticity. This is more marked at times than at others.

ASSOCIATED MOVEMENTS.—In talking or performing voluntary acts with the arms or legs or in attempting to rise from a prone

posture, the whole body seems to take part in irregular movements which are particularly marked in the face and in the arms and hands; these movements are athetoid-like.

ELECTRICAL REACTIONS.—Are normal.

CRANIAL NERVES.—There seems to be no palsy of the third, fourth or sixth, and none of the seventh or fifth nerves. Taste is normal. Sensation in the trigeminal distribution is normal. The optic nerve is normal and is not affected in any way. Hearing is acute, and there does not appear to be any paralysis of the palate, tongue or vocal cords, although it is extremely difficult to examine the throat, on account of the great spasticity and rigidity in the muscles of the mouth, face and throat.

MENTAL STATUS.—The patient is suffering from a real dementia and has reached the level of the low imbecile type. She is extremely emotional, cries and laughs involuntarily, although the elated mood is more frequent and constant. She is, at times, however, very vicious and will scratch and bite. She leads a practically negative vegetative existence.

When in complete rest, the tremor is observable and highly suggestive of the paralysis agitans type in the hands, but when the patient attempts to do something, she uncovers a generalized tremor, particularly marked in the hands and head and trunk, of the intention type. The tremor is, at all times, rhythmical, has moderate force, and not very much amplitude and is very much increased by stress and emotion. The patient seems able, with coaxing, to control the tremor and at times is able to stop it altogether. It is not exactly of the intention type either, for the patient is able to drink out of a cup without spilling a drop. At the commencement of the act of grasping the cup and just before she is able to do this, the tremor is quite marked, but as soon as she grasps the cup and brings it to her lips, the tremor practically stops, and she is able to drink the contents without spilling them.

The roentgen-ray photograph is negative. Special eye examinations are also negative. Fluoroscopic examination of the liver has not been made because this method of examination is not available at the hospital. Blood Wassermann is negative.

Speech Disturbances in Tabes.—Walter B. Swift³³ brings up the subject of defective speech in tabes, which he differentiates from the speech disturbances seen in general paresis. The difficulties seen in tabes Swift ascribes to a "failure in the report to the central nervous system of the positions of the speech mechanism during utterance. In other words, speech in certain utterances demands certain very exact and complicated coördinations in the speech mechanism musculature. Just these exact coördinations are impossible, due to a lack of sensory report of their position."

The speech in tabes which this author describes has a slurring, slovenly, inadequate quality, which vanishes when instructions are given to the patient to talk slowly so that enough time may be given for the report of the positions of the various muscles used in speech to the cortex. The author appears to be a little indefinite in his anatomy, referring the entire matter of coördination and position sense of the speech musculature to the cortex without especially mentioning whether he means the cerebral or the cerebellar cortex. Anatomically, we know that there are two connections, the direct or posterior spino-cerebellar tracts which are affected primarily in Friedreich's ataxia, and secondarily in tabes, and are only a portion of the afferent cerebellar connections of the middle lobe of that organ. An important portion of this pathway is received from the columns of Goll and Burdach, or at least, from their nuclei, and helps, with the direct spino-cerebellar tract and the vestibulo-cerebellar and the olivo-cerebellar connections to make up the inferior cerebellar peduncle, so that not only in tabes, but also in the other conditions already mentioned, speech defects occur where the afferent cerebellar mechanism is disturbed, the position and muscle sense of the speech musculature being interfered with. It is needless to say how frequently, particularly late in the disease, mental defects occur in tabes—the taboparesis of authors.

All of the cases cited above serve to show that the cerebellum plays no little part in the proper coördination of the motor part of speech. It makes for plasticity of speech and for variableness of many of its physical characters.

It helps and no doubt entirely directs the motor coördinations of the speech musculature, and the cerebellar speech mechanism when functioning properly enables man to speak quickly, to change the voice in tone and pitch, to impart emotion to the voice and, in fact, gives it its finer qualities. Is this all the cerebellum does? Following out our very broad conception of the speech mechanism, we feel that this is only a small part of what the cerebellum helps to do. We also feel that there is an anatomical basis for our views. In developing these views, however, we must have reference to our anatomical and physiological knowledge concerning the cerebellum.

In abstracting what we already know of the functions of the cerebellum, and in recounting some of the anatomical points involved, reference is had to the chapter on the physiology of the cerebellum in Starling's³¹ book, and to the fine little work by C. J. Herrick,¹⁰ 1916. With that function of the cerebellum which has to do with the maintenance of equilibrium and other changes in the posture of the body not especially connected with visual and auditory stimuli, namely, the vestibulo-cerebellar mechanism, we are not at all concerned, at least not primarily. With the matter of coördination, however, we are entirely concerned. The coördinating mechanism of the cerebellum depends on the reception of afferent stimuli from the muscles, joints and ligaments of the body; but as we are only discussing speech, only the muscles of speech need be mentioned. This system of afferent nerves has been called (by Sherrington²⁸) the proprioceptive system, because it is not excited by changes in the environment, but only by changes within the body itself. Excitation of this system results in a regulation of the extent of motor discharge in the muscles and prevents over-action even though it may be evoked. It is a "compensatory reflex in an opposite direction to the reflex immediately excited from the skin." In this way, according to Sherrington, tone is maintained in muscles. Most of the speech defects, therefore, seen in tabes and Friedreich's ataxia are due to changes in tone and to disturbances of the regulative coördinative mechanism, resulting in ataxia and various types of dysphonia and dysarthria

of ataxic character. The other proprioceptive part of the cerebellar mechanism, which really does not concern us in the discussion of speech, comes in through the vestibular tracts either directly to the nuclei of the cerebellum, or, first, to the vestibular nuclei and then through the inferior peduncle, to the dentate nucleus. This pathway also is mainly afferent.

The efferent pathway of this proprioceptive system is from the roof nuclei of the cerebellum, particularly the dentate, through the superior peduncles into the decussation of the peduncles which cross just below the roof of the aqueduct of Sylvius at the level of the red nucleus. The red nucleus is the first relay in this neuron; the second relay leaves the red nucleus and crosses soon thereafter, forming by this recrossing the rubro-spinal tracts. The red nucleus, very evidently, therefore, is an important relay in this efferent cerebellar pathway, and has more or less important connections with the cerebrum which are both afferent and efferent, and with the corpus striatum by way of the ansa lenticularis. It is in the destruction by disease of this mechanism that we get the speech disturbances and other symptoms of those two extrapyramidal diseases known as paralysis agitans and Wilson's disease.

More important, however, than all these pathways is the enormous pallio-cerebellar pathway from the large association areas, particularly in the frontal and temporal regions of the cerebrum through the basis pedunculi into the nuclei of the pons, and thence by a second relay to the opposite cortex of the cerebellum. This pathway is known as the middle cerebellar peduncle, and in size is much larger than the area occupied by the pyramidal tracts in this region. The importance of this pathway is tremendous and its functions are shrouded in mystery. That it must be important is evident not only from its size but also by the fact, as given by Starling,³¹ that there is a close association between the development of each cerebral and the opposite cerebellar hemisphere. "Atrophy of one-half of the cerebrum brings about atrophy of the opposite hemisphere of the cerebellum." The tremendous size of the cerebellar cortices which are made up almost entirely (if we exclude the middle lobe of that organ) by this connection, also attests

to the great importance of this pathway. No attempt has been made to go very thoroughly into the physiology or anatomy of this most interesting subject. Only those points which seem to bear on our argument are mentioned. The physiology of this region is also extremely interesting. Experiments by Ferrier⁸ show that relatively much greater stimuli must be applied to the cerebellar cortex in order to get a motor response than is necessary to obtain the same degree of response by stimulation of the cerebral cortex. When such responses do occur, as a result of stimulation of the cerebellar cortex, they are always of limbs and large units of the body, not of single muscles or groups of muscles as when the motor cerebral cortex is stimulated. On the other hand, movements excited by stimuli from the nuclei of the cerebellum are not quite so widespread, and can be initiated with much slighter stimuli than is necessary to produce the same degree of movement when applied to the cerebellar cortex. It seems, therefore, that the functions of the cortex of the cerebellum appear to be different from those of the nuclei of that organ. Horsley regards the cortex of the cerebellum as an afferent receptive center entirely, and the nuclei as efferent centers, which have to do with movements of the eyes and head, trunk and limbs, and are mainly concerned with the maintenance of equilibrium in response to alterations of the positions of the body.

While this gives us very little definite information about the functions of the cortex of the cerebellum, enough has been said to indicate that these functions are but little understood, and that they are for that reason probably of great importance. How important, may be seen from Herrick's¹⁰ discussion of this matter. In correlating the importance of this cerebro-cerebellar pathway, we must turn first to this author's idea of the functions of the cerebral cortex (p. 298): "There is a specific localization of function in the cerebral cortex, in the sense that particular systems of sensory projection fibers terminate in special regions (the sensory projection centers), that from other special regions (the motor projection centers), particular systems of efferent fibers arise for connection with the lower motor centers related to groups of mus-

cles concerned with the bodily movements, and that between these projection centers there are association centers, each of which has fibrous connections of a more or less definite pattern with all other parts of the cortex. The destruction of any part of the cortex or of the fiber tracts connected therewith, involves, first, a permanent loss of the particular functions served by the neurons affected, and, in the second place, a transitory disturbance of the cortical equilibrium as a whole (diaschisis effect of von Monakow). Specific mental acts or faculties are not resident in particular cortical areas, but all conscious processes probably require the discharge of nervous energy throughout extensive regions of the cortex, and the character of the consciousness will depend in each case upon the dynamic pattern of this discharge and the sequence of function of its component systems. This pattern is inconceivably complex and only the grosser features are at present open to observation by experiment and pathological studies.

"No cortical area can properly be described as the exclusive center of a particular function. Such 'centers' are merely nodal points in an exceedingly complex system of neurons which must act as a whole in order to perform any function whatsoever. Their relation to cerebral functions is analogous to that of the railway stations of a big city to traffic, each drawing from the whole city its appropriate share of passengers and freight; and their great clinical value grows out of just this segregation of fibers of like functional systems in a narrow space, and not to any mysterious power of generating psychic or any other special forces of their own.

"The essence of cortical function is correlations, and a cortical center for the performance of a particular function is a physiological absurdity, save in the restricted sense described above."

Again on page 290, speaking of the association centers of the cortex, Herrick¹⁰ says: "These alone are greatly enlarged in the human brain as compared with those of the higher apes. In the latter animals, the projection centers are fully as large as those of man, the much smaller brain weight being chiefly due to the relatively poor development of the association centers." Now we find that "the

cerebral cortex is in direct connection with the red nucleus of the cerebral peduncle by a cortico-rubral tract, arising in the frontal region of the cortex, and by ascending fibers from the red nucleus to the same general part of the cerebral hemisphere. From the frontal, parietal, temporal and occipital association centers there arise large descending fiber tracts to the nuclei of the pons (cortico-pontile tracts). These connections between the cerebral cortex and the red nucleus and pons put the cerebral cortex and the cerebellum into very intimate relations, but the exact way in which the cerebrum and the cerebellum coöperate functionally is obscure."

This pathway by means of the cortico-pontile tracts, or, as they are also called, the middle cerebellar peduncles, between these tremendously important and highly developed cerebral association centers and the mysterious cerebellar cortex, assumes a position of very great importance. In structure, the cerebellar cortex differs from the cerebral cortex in the form and arrangement of its neurons, the former being similar throughout in construction. The cerebral cortex, as is well known, shows marked differentiation in various places in the character, form and arrangement of its cells and neurons. We already know that the functions of the cerebrum are of the highest type, and, together, give us in man that momentous thing which we call the human mind, or intelligence, in the broadest sense. This much seems to be undoubted. The function of the cerebellum, from all its known physiology and undifferentiated anatomical structure, seems to be admirably adapted only to one thing, namely, coördination and the maintenance of equilibrium. Why, therefore, should a different function be ascribed to the cerebellar cortex when considering its relations to the cerebral cortex? Why not the function of coördination, of cerebral mechanisms? Reasoning logically, therefore, if the cerebrum is the seat of the highest functions of intelligence, and if speech is nothing but the expression of this intelligence, then, of course, the function of the cerebellum in relation to the cerebrum is the coördination of the various mechanisms of speech and intelligence,—coördination, for instance, of thought production and the emotions,

the disturbance and dissociation of which in normal states is best seen in dementia precox with all its attendant protean symptomatology. Stewart Paton is very fond of describing the mental defect in dementia precox as an intellectual ataxia, an illustrative phrase which may have in it more truth than convenience. Of course, this is entirely speculative. At least, there are anatomical reasons why this conception may be possible.

In the types of cerebro-cerebellar agenesis of cerebro-cerebellar diplegias involving injuries at childbirth, this conception finds clinical corroboration. These children show a disfunction in all the fields of cerebral and cerebellar mechanisms, namely, of intelligence, motor and sensory disturbances, and gross defects or entire absence of speech. In the heredo-cerebellar ataxias, the same holds true to a more moderate degree. All writers on this subject, not only of Marie's heredo-cerebellar ataxia, but also of Friedreich's ataxia, comment on the regularity with which intelligence defects, amounting in many cases to dementia and imbecility, occur together with emotional and speech defects. In tumors of the cerebellum and of the cerebello-pontile angle, disturbances of intelligence of a profound character are often seen very early. In a great many autopsied cases of dementia precox reported by Southard,²⁹ previously discussed, there were marked changes in the cerebellar cortex and in the nuclei, so much so that Southard postulated a group of dementia precox cases on his cerebellar findings. In the great majority of all these cases, of course, the cerebrum was correspondingly severely involved. The evidence seems to point to a necessary correlation, therefore, of processes in both the cerebrum and the cerebellum, for the production of these grave speech-intelligence and motor-sensory defects seen not only in dementia precox but also in the other conditions above discussed. It is, of course, realized that nothing of a positive character has been contributed to this subject; the matter has consisted entirely of speculation on a clinical and anatomical basis. It is hoped, however, that others better trained than the author will investigate these suggestions with an idea of substantiating or disproving the conceptions herein set forth.

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NEUROLOGICAL BULLETIN

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CYST OF THE INTERPEDUNCULAR SPACE

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The following case is reported in the hope that others may profit from some of its unusual characteristics and be induced to attempt the further development of a therapeutic procedure for what, up to the present time, have seemed to be unapproachable lesions of the brain. An examination of the literature has failed to reveal any case which has been successfully relieved, and yet the technical difficulties do not appear to offer insurmountable obstacles.

S. T., P. H. No. 31417, 18 years of age, male, single, Russian, driver by occupation.

Present Illness. Complaint.—The patient complained of a gradually failing vision of the left eye which was noticed by chance in December, 1915, and increased for several weeks. After examination by an oculist, he was instructed to wear glasses to improve the sight in the right eye, but was told that the left eye could not be improved. He began to have headaches two months after the onset of the visual trouble. At first these headaches were not particularly severe in the morning, but increased until the pain was general throughout the head and continuous night and day. This condition lasted four weeks. Since that time he has had short attacks of headaches lasting fifteen to thirty minutes. He had no associated dizziness, nausea, or ataxia. He has no muscular weakness, and is mentally as alert as ever.

Previous and Family History.—His previous history is negative, nor is there anything of particular moment in his family history.

The patient was seen at the Vanderbilt Clinic on May 4, 1916, and was referred to the Surgical Service of the Presbyterian Hospital as a possible pituitary tumor on the ground that he showed some evidence of having had bitemporal hemianopsia.

NEUROLOGICAL EXAMINATION MADE

JUNE, 1916

In appearance the boy was well nourished, dark skinned and of small stature.

Coördination.—Normal.

Skilled Acts.—Normal.

Gait.—Normal in all phases and to all signs.

REFLEXES:

(a) *Deep.*—

	<i>Right Left</i>	
	Very active	I
Jaw		
Pectoral	I	I
Triceps	I	<I
Biceps	I	<I
Radial	I	<I Spread to triceps
Wrist	I	<I
Ulnar	I	<I
Patellar	I	<I
Achilles	I	<I
Dorso-cuboidal	I	= I
Periosteal	I	= I Both very active
Crossed periosteal	I	= I Both crossed

(b) *Superficial*.—

Ciliospinal	Pupils dilated
Supra-umbilical	1 = 1
Suprapubic	1 = 1
Upper lateral abdominal	1 = 1
Lower lateral abdominal	1 = 1
Cremasteric	1 < 1
Plantar	} Negative
Modifications	
Clonus	None

Muscle Strength.—Good.*Muscle Tone*.—Normal.*Electrical Reactions*.—Normal galvanic.

General Sensory.—No subjective changes, no pain except right frontal headaches which spread rapidly to entire head. No objective sensory changes.

CRANIAL NERVES

Olfactory.—Normal subjectively and objectively.

Optic.—No diplopia. No nystagmus. Motility normal. Dr. Holden reports: Vision R. 3/200; L. bare light perception in nasal half of field. No light perception in temporal field. Right field shows temporal hemianopsia with controlled left field. Right disc, normal; left disc, shows a diffuse pallor. No elevation of either disc.

Oculomotorius, Trochlearis and Abducens Nerves.—Normal.

Facial and Trigeminal Nerves.—Normal. Corneal sensibility, normal.

Glossopharyngeal, Vagus, Spinal Accessory and Hypoglossal Nerves.—Normal.

Acoustic Nerve.—Cochlear division is normal objectively and subjectively to all tests on both sides. Vestibular division of both sides is normal to galvanic, caloric and rotation tests.

Rectal, Vesical and Sexual Control.—Normal.

Mental Condition and X-ray.—Normal.

Laboratory Tests.—All negative.

At this time the diagnosis of a brain tumor was made. The tumor was located in the basal region of the brain, involving

the left half of the chiasm, the left optic nerve, and the left optic tract.

On account of the apparently inaccessible location of the lesion, the patient was advised to return home and report from time to time for further observation.

The patient was discharged from the hospital June 21, 1916.

His follow-up history contains notes made on the following dates: October 29, 1916, April 15, 1917, October 13, 1917, and January 7, 1918.

On October 29, 1916, he reported no change in eye-sight. He had been free from headache since leaving the hospital and had not suffered from dizziness, vomiting or nausea, nor had he gained or lost weight. He had been employed in a grocery store.

On April 15, 1917, he reported that he was still working in the same position. He had no headaches and his appetite was good. He still had complete blindness of the left eye and temporal hemianopsia in the right eye.

The notes made on October 13, 1917 and January 7, 1918, were essentially the same as these above quoted.

About the middle of February, 1918, he began to notice a weakness in his right arm and leg. Gradually this weakness increased until it became quite difficult for the patient to move his right arm and leg. He was able to control the movements of the leg better than those of the arm. In six weeks' time the difficulty in movement had become so pronounced that all purposeful movements of the arm were impossible. At times he had sensations as if the right leg were asleep. The vision in his left eye had not improved and vision in the right eye was failing.

Upon examination in April, 1918, he showed the following:

VOLUNTARY MOTOR SYSTEM

Abnormal Attitudes and Deformities.—There are no deformities, although the right hand is held in the hemiplegic attitude during locomotion.

Gait.—He presents a definitely hemiplegic gait with defect on the right side, represented by a swinging of the leg and shuffling of the right foot. Hemiplegic exaggeration of automatic associated move-

Scapular	3	2
Suprapatellar	5	3
Patellar	4	3
Achilles	5	3
Dorso-cuboidal	3	2
Periosteal	3	0

(b) *Superficial.*—

Ciliospinal	2	1
Supra-umbilical	1	1
Suprapubic	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1
Cremasteric	2	1
Babinski	2	0
Chaddock	2	0
Oppenheim	2	0
Gordon	0	0
Schäfer	0	0

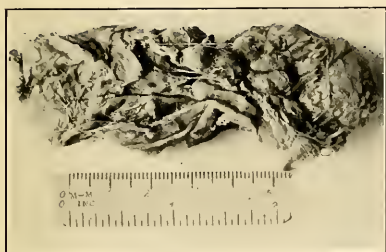


FIG. 1. CYST REMOVED FROM INTERPEDUNCULAR SPACE.

ments are present in the right arm and hand. He walks better on the left than on the right foot, and no changes are observed in gait when the patient walks with the eyes closed. All tests for the gait revealed the right hemiplegic defect.

Coördination.—Equilibratory coördination is normal. Romberg's sign is negative. He stands better on the left than on the right foot. Non-equilibratory coördination is normal except in so far as the movements of the right arm and leg are disturbed by paresis.

There was no evidence of adiadochokinesis or dysmetria.

Skilled Acts.—No apraxia or aphasia was present.

REFLEXES:

(a) *Deep.*—

	Right	Left
Jaw	1	3
Pectoral	3	2
Biceps	3	2
Triceps	3	2
Radial	3	2
Ulnar	3	2
Wrist	3	2 to triceps on R.

Abnormal Involuntary Movements.—No tremors, twitchings, athetoses, spasms or convulsions were reported or observed.

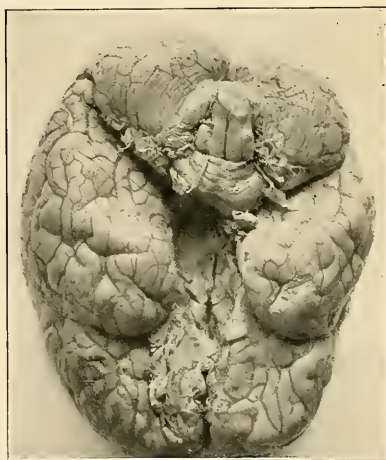


FIG. 2. SHOWING BED OF CYST IN INTERPEDUNCULAR SPACE.

Muscle Strength.—Patient coöperated well and showed marked paresis of right arm. The weakness was more marked in the extensor than in the flexor groups.

Muscle Status.—No change in contour of the upper extremities, but a slight atrophy was evident in the right thigh and leg. There were normal consistency and hypertonus of the right leg and arm, and a slight increase in muscular irritability in the muscles of the right arm and leg. There was no myo-edema.

Abnormal Associated Movements.—The following movements of this variety were present on the right side:

1. Radialis sign of Strümpell.
2. Raimiste's sign of flaccidity.
3. Neri's sign.
4. Babinski's trunk-thigh sign.
5. Grasset-Bychowski's bilateral leg sign.
6. Grasset-Gaussel's sign and Hoover's complementary opposition sign.
7. Raimiste's adduction and abduction sign.
8. Strümpell's tibialis sign.
9. Marie-Foix's tensor vaginae femoris sign.
10. Huntington's coughing sign.

Nerve Status.—Normal.

General Sensory.—All phases, modalities and qualities of somatesthetic sensibility were normal.

CRANIAL NERVES

1. *Olfactory.*—Normal subjectively and objectively.

2. *Optic Nerve and Oculomotor Apparatus.*

Vision.—Left eye showed complete amaurosis. Right eye, failing vision.

Eye grounds showed a double optic atrophy. Both pupils were irregular, the left being larger than the right. No hippus was observed.

The right pupil reacted to light directly, but the left pupil did not. There was no consensual reaction from right to left, but there was a slight reaction from left to right. The left pupil did not react to convergence or accommodation, but the right pupil was normal. Both palpebral fissures were normal. There was no exophthalmos, enophthalmus or strabismus. Conjugated eye movements were normal.

Nystagmus occurred, when the eyes were directed upward, there was slow movement to the right. Directed to the right the eyes showed a slow movement to the left.

Winking and blinking reflexes were present on the right, but absent on the left.

3. *Auditory Nerve.*—Normal in both divisions. Bárány caloric test gave normal results from both ears (typical labyrinthine nystagmus). Nausea was produced by test from right ear.

4. *Trigeminus Nerve.*—Jaw jerk was more active on the right side. Conjunctival and corneal reflexes were present on both right and left sides. Supraorbital reflexes of McCarthy were present and active on both sides.

5. *Facial Nerve.*—Paralysis of the right lower facial muscles.

6. *Glossopharyngeus, vagus, spinal accessory and hypoglossal nerves* were all normal.

Mental Status.—Normal.

Systemic Status.—There was a bronze pigmentation of the trunk which was well marked. This ceased at Poupart's ligament, leaving his lower extremities unpigmented. In general, the nodes were enlarged.

Laboratory Tests and Roentgen Ray.—Negative.

Upon these findings the former diagnosis, tumor of interpeduncular space, was considered confirmed.

OPERATION, APRIL 27, 1918

The anesthetic was intrapharyngeal ether.

Incisions.—1. Transverse, parallel to supraorbital margins from just above one angular process to the other. 2. Median, from glabella to 5 cm. posterior to bregma. 3. Transverse from just above one ear across the vault 5 cm. behind bregma to just above opposite ear.

There was considerable intracranial pressure causing brain to bulge. The dura and pia were thickened and had a whitish opaque appearance.

With Hudson's drill and Gigli's saw a rectangular bone flap was removed from the left side, having a pedicle through which

it was fractured corresponding to the squamous portion of the temporal and lower portion of the parietal bones. The dura was then opened and an attempt made to raise the frontal lobe from the orbital plate to obtain a view of the chiasm. On account of the extreme in-

The patient's condition became suddenly worse, and it was decided to postpone any further procedure. His pulse became rapid; he became cyanotic, and his breathing was rapid and labored. The wound was closed with interrupted silk suture; the patient was given a saline infusion of 500 c.c. without benefit, and returned to ward in profound shock. His temperature rose at once to 105°, pulse 140. Four hundred cubic centimeters of blood were given by transfusion without effect.

The patient died five hours after operation, never having regained consciousness.

AUTOPSY FINDINGS IN THE BRAIN

In general appearance the brain before removal from the skull seemed normal. The dura mater, except for evidence of operation, showed nothing pathological. The pia mater was much injected over the operative fields, but there were no extensive intrapial or subpial hemorrhages. The arachnoid was normal. Upon removal from the skull, the convolutions and fissures of the vertex and lateral aspect of either hemisphere showed no notable changes in any area.

The basal surface showed a large cyst, 8.5 cm. antero-posterior diameter, 3 cm. posteriorly, 3.5 cm. anteriorly, and a constricted portion 2 cm. midway between the anterior and posterior poles (Fig. 1). The cyst was filled with a clear, limpid fluid whose walls showed some areas of thickening of a white, chalky appearance. There were numerous blood-vessels, some of considerable size, upon the surface. The cyst impinged posteriorly upon the right side of the cephalic border of the pons immediately anterior to the exit of the trigeminal nerve. It extended forward between the mesial aspects of the poles of the two temporal lobes, reaching forward across the inferior of the left frontal lobe so as to displace the right orbital surface of the frontal lobe to the right and the left orbital surface upward and to the left. The anterior pole of either

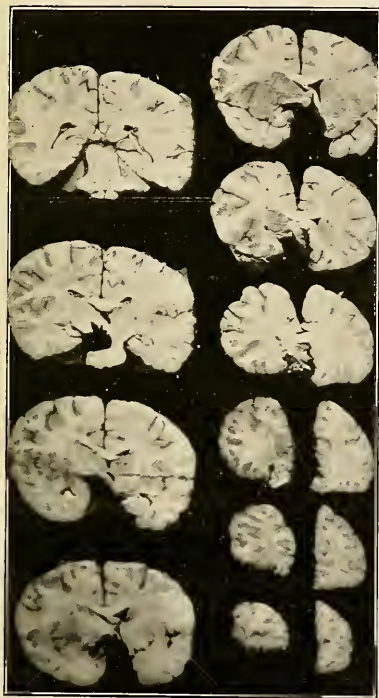


FIG. 3. MACROTOMIC SECTIONS OF BRAIN SHOWING RESULTS OF PRESSURE OF CYST.

tracranial tension this was not possible, and it was decided to turn back a similar bone flap on the opposite side, expose the opposite frontal lobe, and thus obtain room for displacement of the brain. This was accomplished without great difficulty, the area over the superior longitudinal sinus being carefully rongeured away without opening that sinus.

frontal lobe appeared normal as did the temporo-sphenoidal lobes. The cerebellum and medulla showed nothing abnormal. (Fig. 2.)

Upon removal of the cyst, its bed was found to comprise the following from behind forward:

1. The anterior transverse fibers of the pons were displaced slightly backward on the left, showing some signs of pressure and resulting in a partial rotation of the pons upon the vertical axis.

2. The left cerebral peduncle was compressed and displaced toward the left, thus widening the interpeduncular space.

but slightly compressed and displaced to the right. The tract, however, was markedly flattened as it crossed the peduncle and the optic nerve remained simply as a small fibrous band.

Upon macrotomic section the degree to which the basal structures of the brain had been compressed was more clearly demonstrated. A series of such sections is shown in Fig. 3.

The more detailed results of the compression are shown in Figs. 4 and 5.

Figure 4 reveals to what extent the left internal capsules had been compromised and this explains the right

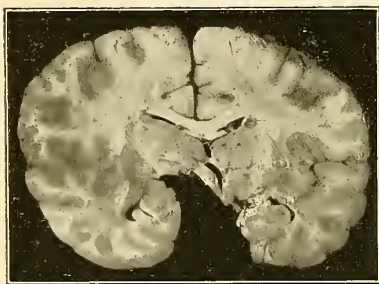


FIG. 4. SHOWING DETAILS OF STRUCTURES COMPRESSED BY THE CYST.

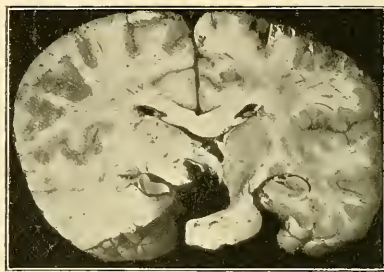


FIG. 5. SHOWING DETAILS OF STRUCTURES COMPRESSED BY THE CYST.

3. The right cerebral peduncle appeared but slightly affected by the pressure.

4. The pituitary body, together with the tuber cinereum, was pushed outward, backward and to the right, being situated in the angle between the cephalic border of the pons and the right uncus. The pituitary body showed no changes in the gross.

5. Both mammillary bodies were displaced to the right and flattened.

6. The post-chiasmatic eminence was thinned out as a diaphragm, measuring 2.5 cm. antero-posteriorly by 2 cm. transversely.

7. The chiasm remained as an atrophic band from the pressure of the cyst.

8. The right optic tract and nerve were

hemiplegia with the increased and pathological deep reflexes upon the right side, together with the right Babinski. This also affords the explanation of the appearance of the several abnormal associated movements and the hypertonus on the right side.

The lenticular nucleus and thalamus show considerable compression, a fact which is of some interest since clinical examination disclosed no symptoms referable to disturbances in these parts of the brain.

The dysaesthesia of which the patient complained in his right leg was doubtless due to capsular compression. The absence of more marked oculomotor disorders than the patient manifested may be attributed

to the manner in which the oculomotor nerves were affected. With the exception of their more cephalic fibers both nerves had escaped serious involvement by the cyst, having been slowly pushed to the sides. On the other hand, certain of the oculomotor symptoms must be taken to indicate a slight degree of third nerve involvement, as for example the nystagmoid movements observed in the patient; these represent a muscular imbalance rather than a true nystagmic disorder. The defective light and convergence reflexes almost certainly are dependent upon the pressure from the cyst which affects the more cephalic fibers of the third nerve.

DISCUSSION OF DR. LAMBERT'S CASE

DR. O. S. STRONG.—I was impressed by the fact that with the third nerve there was, apparently, only a lack of reaction to convergence and light. The root fibers to the internal eye muscles lie in the more cephalic part of the nerve root, and consequently would be more affected by such a lesion. Only section could show how much is intact. As far as absence of sensory changes go, there again you cannot furnish complete explanation without sections.

DR. WARD A. HOLDEN.—This case has a clinical interest for the ophthalmologist. We have been inclined to distinguish between pituitary tumors and these cysts by the position of the sella turcica as shown by the roentgen ray. In true pituitary tumor which causes the same visual field symptom, the fossa is practically always enlarged. In the past before these operations were so commonly done, I think all of us saw a number of cases with no enlargement of the pituitary fossa, but with symptoms indicating pressure on the chiasm, where the patients eventually went blind. Those symptoms we supposed to be due to higher cysts. Cushing seemed hopeless of the advisability of operating on such cases because of the possibility of recurrence.

DR. MARK J. SCHOENBERG.—Regarding the examination for determining involvement of the third nerve, I should like to make a reservation as to the diagnosis that the third was not involved. The patient was blind in the left eye, and you could not elicit diplopia under such circumstances. So if you wanted to test the left eye, the only way was to tell the patient to look this way, and he did not see. This test for the action of the fibers of the third nerve in the left eye is unsuitable. So there may have been involvement of the third nerve on the left side without its being possible to elicit it.

There is another thing to be considered. Walker of Boston, of Cushing's clinic, wrote an admirable article regarding these tumors and their effect produced by pressure on the chiasm. He is doubtful from the postmortem examination of a number of cases whether the atrophy is not due rather to traction than to pressure. He believes atrophy results more often from traction than from pressure. He has seen cases where pressure which was exerted for a long time, between twelve and fifteen months, was not sufficient to produce complete optic atrophy so as to destroy visual function. This shows that the optic nerve can stand a good deal of pressure.

The question comes up then, why should this be on the third nerve? Suppose he did not have involvement of the third nerve; it may have been pushed out of the way, and conditions might have been such as not to destroy its functions. But I believe the third nerve was involved, and it was not possible to elicit it.

I should like to ask if Dr. Lambert considers the test for sugar tolerance in pituitary conditions a reliable one.

DR. S. PHILIP GOODHART.—This very instructive case, so carefully observed and fully reported, brings up several interesting problems. Not the least of these is the relation of pressure of a neoplasm of the brain upon important nerve structures to the loss of function of these centers. It is often

difficult to reconcile the comparatively slight disturbance of function with the apparent extensive destruction of nerve tissue. One sometimes sees a large growth occupying a considerable portion of a hemisphere apparently disturbing recognized centers, and yet the clinical picture may have been relatively slight. Evidently several factors determine this. The present case shows a large cystic mass at the base, and one would have expected from the anatomical extent and from the appearance of displacement as well as from gross appearance, that the left olfactory and left oculomotor nerves would have been completely paralyzed. Clinically, it appears that the former was unaffected and that the latter was only in small degree affected. I think the third nerve was really involved, as shown from the failure of consensual contraction to light of the left pupil when the right was stimulated. May not the character of the growth be the cause of the slight degree of functional loss? A cyst is of course flexible, and its pressure not as constant and unyielding as one of the solid tumors.

I have wondered, since tumors that press upon the pituitary body seem to give much the same picture as those that really emanate from that structure, whether it could not be said that in the former the characteristic metabolic changes are absent or appear only very late. One would expect that early histopathologic changes in tumors of the gland itself would occur early if the posterior lobe were affected. I doubt the value of sella roentgenograms in these cases, though they occasionally do help us; but in my experience they are uncertain, and not convincing.

The conservative operative measure in basal tumors seems to be a frontal decompression, though Dr. Lambert's skillful effort to remove or evacuate the cyst was almost successful. I recall Dr. S. P. Kramer's advocacy of frontal decompression for basal tumors. He claims that more room for forward movement of the brain and greater relief from pressure

are afforded than by the usual temporal decompression.

DR. ADRIAN V. S. LAMBERT.—I think the criticism of the third nerve function and the accuracy of our observations are just. With a proposition as difficult to deal with as third nerve function in a blind eye with slight reaction, I think we might have missed it. It seems to me in looking at the brain that this nerve is involved.

I do not believe sugar tolerance and metabolic study are very valuable in determining the possibility of involvement of the pituitary. Dr. Holden's point as to the roentgen ray being more valuable is a good one. But this patient was only eighteen years of age, and my experience has been that the pituitary fossa in these young skulls does not show up well in the roentgenogram, particularly the posterior boundaries. I saw one case in a boy of fifteen with a large tumor of the pituitary that had risen posteriorly and grown backward, and we thought it was a cyst not of pituitary involvement, on account of lack of evidence of absorption of the confines of the pituitary fossa. The bones are soft, and there is too great lack of development of bony structure to give a good shadow. In the adult the roentgen ray is a more valuable criterion than sugar tolerance.

In looking over this brain I was surprised at the size of the cyst and the degree of destruction of structures which would seem to be among those which, if involved, should give definite clinical symptoms. Take the third nerve, squeezed, observe how flat it is and compare it to those nerves freed from exuberant callus. The nerves here in this brain are very much more distorted than in cases of paralysis associated with callus; and I shall be surprised if they do not show considerable changes on section, more than those nerves which give clinical symptoms of a pronounced character following exuberant callus.

What struck me were the great changes referable to the peduncle that took place

in the boy's condition within a year. He had extended backward in its growth. developed distinct hemiplegia in one year, I should like to try another case from the though showing no sign of it before, which therapeutic standpoint, for I believe that would make it appear that the cyst had it is possible to expose the region of the increased rapidly and, starting anteriorly, optic chiasm through a frontal approach.

THE MECHANISM OF PAIN FROM THE PHYSIOLOGICAL STANDPOINT

PRELIMINARY NOTE

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Pain has been aptly described by Sherrington as the psychic equivalent of the nociceptive reflex. The theory proposed is based on clinicopathological studies embracing lesions of the nervous system at various levels from the periphery to the cerebral cortex. The author regards the afferent nociceptive arcs as the forerunners of the pain paths leading to the optic thalamus, from which indeed these latter are to be considered as developed. All forms of sensibility may ultimately be reduced to two primary forms, viz., (1) affective, and (2) critical. Affective sensibility is that through which we become aware of pain, pleasure or change of state; critical sensibility on the other hand implies comparison. The impulses mediating the fundamental gross affective element (pain or hurt) enter into consciousness mainly in the optic thalamus, whereas the impulses mediating critical sensibility enter into consciousness mainly at a higher level, presumably in the cerebral cortex. The writer rejects Head's division of sensibility in the peripheral system of nerves into epicritic, protopathic, and deep, and offers his own classification as follows:

- (1) Superficial critical sensibility;
- (2) Superficial affective;
- (3) Deep critical;
- (4) Deep affective.

This classification is simpler than Head's or Sherrington's (exteroceptive, proprioceptive, and enteroceptive), and more in accord with the needs of clinical neurology. Superficial critical sensibility is evoked by such stimuli as light touch, degrees of heat and cold ranging about the neutral point or skin temperature, and compass

points simultaneously but lightly applied. Superficial affective sensibility is evoked by such stimuli as pin prick under moderate pressure, cold ranging from 22°C. to 0°C., and heat ranging from 40°C. to 55°C. Deep critical sensibility embraces pressure-touch and its localization, posture and passive movement, size, shape, weight (passive), the compass points simultaneously applied with firm pressure, etc. Deep affective sensibility embraces pressure-pain (pinching, etc.), and heat and cold in extreme degrees, e.g., 0°C. and 55°C. in massive prolonged application.

Under normal conditions areal stimuli make their appeal to both systems of nerves mediating the two great fundamental divisions of sensibility, viz., the affective and the critical. Thus when a hot object touches the skin three distinct elements may be potentially differentiated, viz., touch, warmth and hurt. So, too, when a cold object touches the skin one may differentiate potentially between touch, cold and discomfort depending on circumstances. In pin prick also touch sharpness and hurt may be differentiated potentially. The writer insists that for practical purposes the sensations evoked by prick and by gross heat and cold must be regarded as analyzable into affective and specific critical elements, the critical element of prick being sharpness, that of heat being the specific sensation of heat (warmth), and that of cold the specific sensation of cold. The fundamental affective elements of each of these stimuli is the same, viz., hurt or discomfort. The writer insists on these distinctions, because of the dissociation of sensibility observed

by him in lesions of the nervous system at various levels, viz., in the peripheral nerves, in the posterior nerve roots, in the spinal cord, in the brain-stem, in the optic thalamus, in the internal capsule, and in the cerebral cortex. At each of these levels dissociation was observed in which the fundamental or hurt element was more or less sharply differentiated from the critical elements. In fact, it is upon such a dissociation, encountered in lesions at the various levels mentioned, that his theory is mainly founded.

Head and his colleagues first called attention to a peculiar type of sensibility which appeared about eighty days after suture of a divided nerve. Affective stimuli were adequate for this type of sensibility, which was further characterized by over-reaction, wide radiation, reference, poor localization, and inability to appreciate the nature of the stimulus. This type of sensibility, which Head called protopathic, appeared long before the other type, to which he gave the name epicritic, and for which the adequate stimuli were such as those already set down under the heading of superficial critical sensibility. Both the observations of Head and his conclusions have been challenged by subsequent observers; but the writer's experience lends material support to Head's contentions in all essential respects. The fact already noted by Head, that as critical sensibility is gradually restored the extremely disagreeable hyperalgesia characteristic of protopathic sensibility gradually disappears, shows clearly that in some way the mechanisms mediating critical sensibility exert a controlling, restrictive influence upon the mechanisms mediating the hurt or pain elements, and furnishes a clue to the mechanisms responsible for the extraordinary sensory phenomena witnessed in lesions of the ventrolateral aspect of the thalamus, wherein is encountered dissociated sensibility closely resembling, if not identical with, that found in the regeneration period after nerve section and suture.

Head and his colleagues did not believe that lesions of the medulla spinalis (spinal cord) could cause dissociated sensibility like that seen after nerve section and suture; but in certain cases of tumor, in which the medulla spinalis was compressed from the outside, it was possible at certain stages of the compression to demonstrate the existence of this type of dissociation in which the critical elements were suppressed, wholly or partially, while sensibility was retained for affective stimuli, these latter, when felt, being attended by over-reaction, radiation, reference, poor localization, and inability to name the nature of the stimulus. On the other hand, in cases of syringomyelia, the dissociation encountered was such that only the fundamental purely affective or hurt elements in prick and gross heat and cold were suppressed, while the specific critical elements, such as sharpness, cold and warmth, were retained.

In conditions of pathological pain and tenderness, the mechanism immediately responsible for the painful impulses coursing inward from the periphery, resides chiefly in the neuron bodies situated in the dorsal root ganglia. This is apparent from the fact that in lesions of the peripheral nerves such as cause severe neuralgias (tic douloureux), or hyperalgesia (causalgia), section of the nerve distal or proximal to the lesion does not relieve the pain, whereas section of the posterior nerve roots abolishes it permanently. Lesions severing the pain paths anywhere proximal to the posterior root ganglia also abolish pain, whereas those which merely obstruct such pathways tend to diminish conduction, and to this extent are not directly causative of pain, although they may be an indirect cause by rousing the mechanisms in the posterior root ganglia to metabolic over-activity, the latter being the immediate biological response of the affective neurons to anatomical or functional interruption.

The point at which the critical system impinges upon the affective system of

neurons is located within the thalamus, and it is here that restrictive control is exerted by the critical upon the affective system. In brief, then, the theory of the mechanism of pain is as follows: In normal sensibility, the critical system controls the affective system of neurons in such a way that the anabolic and catabolic activities in the affective neurons are nicely adjusted to the needs of circumstance by supplying suitable threshold, etc. The materials requisite for the initiation and conduction of nerve impulses (kinetoplasm, etc.), are supplied in such a manner and to such an extent as to meet in the best manner possible the requisite needs of normal sensibility. Lesions causing dissociation of sensibility, such as those found in the peripheral nerves, in the medulla spinalis, in the brain stem, or in the thalamus, upset these nicely adjusted mechanisms by interfering with the control normally exerted by the critical system upon the affective system. The immediate effect of such lesions is the appearance of pathological tenderness and

hyperalgesia. In all such lesions the affective neurons themselves are directly implicated anatomically or functionally. This interference acts as a stimulus inciting the remaining portions of the affective neurons, and especially the neuron bodies in the dorsal root ganglia, to hypermetabolism in the interests of restoration of functional and anatomical continuity. One of the by-results of such hypermetabolism is the spontaneous or readily elicited overflow of neural energy (nerve impulses) brainwards. This is the cardinal feature of pathological tenderness and spontaneous pains. These two principles underlie the theory of the mechanism of pathological pain and tenderness, viz., (1) dissociation, functional or anatomical, of the critical from the affective system of neurons, and (2) hypermetabolism of the injured or liberated affective neurons. The pain and tenderness in themselves represent a regressive type of sensibility serving a purpose similar to that of such primitive protective mechanisms as the nociceptive reflex.

THE MALONEY METHOD IN THE TREATMENT OF TIC

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A tic is a coördinated purposeful movement. It is in the first instance provoked by some external cause, or by a mental state or process to which it is a more or less adequate motor response. Frequent repetition makes the movement habitual, and leads to its reproduction, without evident cause or purpose, in exaggerated form and frequency. The reproduction of this motor response is often preceded by desire which is so intense as to provoke discomfort if repressed, but which when fulfilled by a tic movement conduces to a feeling of satisfaction. Volitional effort or distraction diminishes its activity, and it disappears in sleep.

There are two types of tic: the clonic and the tonic. The clonic tic is rapid, frequent and intense; it occurs at irregular intervals, and is always coördinated. The tonic tic is characterized by its prolonged duration. Tic may occur at any period of life except infancy. It is especially common at puberty. There is little difference in its incidence in the two sexes. Psychical predisposition, trauma, infectious diseases or mimicry may be blamable causes. Heredity is an important factor; a neuro-pathic or psychopathic family history may often be elicited. I have now under observation a family in which two sisters are tiquers, a third sister suffers from migraine, and a brother has dementia precox; the mother is highly neurotic and the father died of diabetes with which was associated a polyneuritis. Another family is also under observation, where the father, son and daughter are tiquers. Occasionally a family history of alcoholism or syphilis may be obtained. In the subjects of tic and in their families, mental instability

and intellectual superiority have repeatedly been conjoined.

PATHOLOGY.—Tic is a psychoneurosis, and the pathology is unknown. As is the case with many neuroses and psychoses, we must for the present rest satisfied to observe symptoms; their morbid anatomy will remain unsolved until our methods of investigation obtain perfection, or our knowledge concerning functional centers is more precise.

VARIETY OF TICS.—A tic may involve any group or groups of muscles. There may be a number of muscle groups affected at the same time. Blinking tics of the eyelids are most frequently seen; they may be either of the clonic or tonic type, involving one eye or both. Occasionally both types are present in the same patient. Associated with tic of the eyelids we may find sniffing tic of the nose, facial grimaces, and wrinkling of the forehead. Tic of the extrinsic muscles of the eye is occasionally encountered; the eyeballs may deviate to the right or to the left, or they may be moved up and down. Tic of the muscles of the neck is not uncommon. It may be either of the clonic or tonic type; the clonic type may assume a nodding, tossing, or a salutatory character. The tonic type can usually be corrected by some simple device or manœuvre, occasionally of the patient's own invention; it should be unconnected with muscular, osseous or articular lesions of the neck. Tic of the shoulder may affect one or both sides. It may assume a shrugging character, or may be a simple forward, backward, upward or downward jerking of the shoulder. They are generally associated with

neck tics, particularly mental torticollis. Tics of the trunk and lower extremities are infrequent. One of the most habitual tics of the lower extremities is a "kicking tic." Leaping tics are met with occasionally. Of special interest are the respiratory tics, the snorting, sniffing, blowing, whistling, coughing, hiccoughing and sobbing tics, and the correlated speech tics. Respiratory tics may be either inspiratory or expiratory; they are dependent on involvement of the diaphragm or muscles of respiration. Tics of speech are commonly associated with tics of the face or neck. They are caused by the movement of the air through a more or less contracted glottis associated with spasmodic contraction of the diaphragm. Other tics of speech are seen in the form of abrupt, impetuous, irresistible ejaculation of obscene words or curses (coprolalia), or in the form of repeating words or phrases just heard, or spoken by themselves (echolalia). The patient is conscious of the impropriety of the act, but is incapable of resisting the impulse.

DIAGNOSIS.—Difficulty may sometimes arise in differentiating between facial spasm and tic. In spasm the movements occur with extreme abruptness and simulate the contractions produced by electrical stimulation; there is no purposive or coördinated features in spasm, which is usually confined to the facial nerve area. Volition, attention, distraction and emotion fail to effect marked modification of the spasm. No irresistible impulse precedes its manifestation, nor is it succeeded by a feeling of satisfaction. As a general rule the patient's mental state does not present the anomalies met with so frequently among those who tic. Spasm may persist in sleep.

Chorea and tic may have in common a neurotic family history, or a physical or mental crisis preceding its onset. The movements in chorea are more rapid, more irregular; they are more or less incoördinate, and are usually devoid of outward

purpose; observation of the patient usually increases the movements of chorea and diminishes those of tic. The tic movement is more regular, purposeful, periodic and controllable.

PROGNOSIS.—The prognosis of tic varies with the age and mental capacity of the individual and the method of treatment used. Meige and Feindel hold that "the individual can be cured if he can will to be cured." Temporary remissions may occur and should not be mistaken for a cure. Displacement of the location of tic is frequent. Children may, under the influence of training and correction, at times under the influence of threats, drop tic habits. If there is a marked mental inferiority, or mental infantilism, the prognosis is bad, even with improved methods of treatment. The disease is usually chronic, runs a course of months or years, and not infrequently persists throughout the entire life of the individual.

HYGIENIC TREATMENT.—The home surroundings should be made as pleasant as possible; if a source of imitation, such as tic in the parents or other members of the family is present, the patient's contact with the mimical person should be limited. With the exception of a few protracted cases it is usually unnecessary to send tic patients away from their homes. A strict attendant is of decided advantage. The diet should be bland; it should not contain any substances that are likely to increase the excitability of the peripheral nervous system. Coffee, tea and, in adults, alcohol and tobacco should be restricted. Hydrotherapy in the form of tepid douches or spinal drip, is of great benefit.

SURGICAL TREATMENT.—Section or resection of the nerves, particularly of the spinal accessory, has been advocated, but it leads merely to transient or permanent paralysis of the muscles. Mental torticollis is a psychical disease far from the province of surgery. Kocher's plan of cutting successively the muscles involved in the

spasm gives inconsistent and unsatisfactory results. Injection of a mixture of paraffin in oil which would solidify at 37° C. has been tried; comment is unnecessary. The injection of alcohol is said to have been attended with success in a small percentage of cases.

HYPNOSIS.—This has favorably influenced some cases; its application, however, is limited. Psychoanalysis has also been applied, but few cures have been recorded.

REÉDUCATION.—Methods in reéducation have been evolved by various men. These methods are based upon the recognition between the convulsion and mental state of the patient. Oppenheim calls his method "inhibition gymnastics"; Brissaud, Meige and Feindel, "psychomotor-reéducation." Pitres has devised breathing gymnastics. Oppenheim's method consists in training the patient to inhibit reflex, defence and emotional reflex movements. Thus, he takes a pointed instrument and slowly approaches the cornea while admonishing the patient not to blink; or the mucous membranes of the nose are tickled, and the patient admonished to suppress the reflex. Again, physical pain or tickling is resorted to with the command not to draw away the part which is prodded, or not to yield to the impulse to laugh or respond in any way to the tickling. These exercises are all intended to bring the will into play, and to strengthen the power of inhibition and suppression of reflex movements. The method involves regularity of routine and painstaking perseverance.

The method of Brissaud, amplified and extended by Meige and Feindel, consists of a combination of immobilization of movements with movements of immobilization. The patient is taught to preserve immobility; he is taught to replace an incorrect movement by a normal one. The exercises must be graduated at first, and short in duration. The patient is encouraged, and required to remain absolutely motionless, as if sitting or standing for a photograph, first for a few seconds,

then for a longer period of time, up to a half hour. When he can remain immobile for a period of time in these positions, the attitude of the head, arms and legs is varied. Eventually he will learn to maintain immobility of certain parts of his body while he is walking or while he is executing certain movements with his arms.

Hartenburg advises the use of the faradic current to produce the antagonistic movements, the method of overcoming passive resistance to the opposing muscles. In all cases care and supervision are necessary to prevent the antagonistic movement itself from becoming a tic. Each tic must be studied, and treated by special measures which the physician must elaborate and order.

Pitres' method of deep inspiration for the cure of tic is as follows: Supported against a wall, with shoulders braced back, the patient is instructed to take slow and deep inspirations, raising his arms the while, and letting them fall during the expiration. This performance is repeated three times a day for ten minutes at a time. In a modification of this method the patient recites aloud for part of the time. The object is the same as in the other methods, viz., to fix the attention on the allotted task, and to suppress the tic by distracting the attention to some other task. The success of the exercise in individual cases depends upon the ability of the patient to concentrate his attention, and the degree to which he can do it.

Games and sports which demand attention, skill, accuracy and decision should be prescribed. Manual training is of great value, and can readily be made of service through the departments of manual training which are a part of most public school systems today.

In the treatment of tic, part or all of the above described methods of reéducation are useful. Treatment in all cases should begin by teaching the patient the "relaxation or rest exercises" outlined by Dr. Wm. J. M. A. Maloney in his work on the cure of tabetic ataxia. The abdomi-

nal type of breathing is used; the patient is asked to take a deep breath, using his diaphragm, and restricting his thoracic movements, and at the height of inspiration to pause; then slowly and evenly to expire, and again pause. This deep breathing soon tires the patient; so after ten or twelve deep inspirations have been taken, the depth of inspiration and the pauses are shortened until the patient is breathing, without effort, as in sleep.

To relax the muscles, passive movements, in which the muscles are alternately lengthened and shortened, are employed. The muscles of the forehead, cheek and jaw are thus manipulated until the wrinkling of the forehead and blinking of the eyelids disappears, and the muscular spasm is eliminated. Next, a shoulder is relaxed, then an arm; each in turn must be passively moved until all traces of muscular tension vanish and the part lies motionless and flaccid, and falls limply from any unsupported position. The leg on the same side should next be taken. After one part is relaxed, those previously and that newly relaxed should be briefly dealt with again, in the order in which they were first relaxed. This linking of parts previously relaxed is helpful in bringing the whole to a satisfactory state of relaxation. During the passive movements, the operator should continually direct the patient's attention to the control of the chronic twitchings; gradually this requires less and less effort, and soon complete and perfect relaxation is possible.

To relax properly requires continued attention. Relaxation overcomes the fatigue which is invariably induced when these patients attempt to acquire control of the tic movements. The patient in a relaxed state immediately becomes vividly aware of the tic movement, which is under ordinary circumstances performed semi-consciously. The more conscious a movement the more readily is it brought under the control of the will.

When the patient has mastered the rest exercises thoroughly, active movements may be begun. At first simple movements, flexion, extension, adduction and abduction at the joints should be used. These movements should be guided along the normal planes; the rhythm rate and force of the movements should be regulated by counting or using a metronome. Next, the patient resists the movement in order to increase her ability to maintain tonic contraction; and finally the movement is done against the resistance of the operator. When these simple movements have been mastered, more complicated ones are devised. Several joints can be used in complicating the movement. At the same time the patient's attention should be directed to the control of the muscles in which the tic is located. Exercises for these muscles should be ordered and persisted in, until the patient has regained complete control over the muscles involved in the tic.

The psychotherapy of tic is the carrying out of the plans which have been laid down in the above paragraphs. It requires that a definite plan of treatment and of movement be clearly formulated in the mind of the physician, and that the necessity and, above all, the reasons for the plan be conveyed to and impressed upon the mind of the patient; that the latter be convinced of the utility of the treatment, and be incited to the enthusiasm necessary to develop patience and perseverance, without which success cannot be obtained. It consists in influencing the mind day after day, week after week, in such a manner that the exercises will be executed each day with the same care and precision as they were in the beginning. Complete success can be obtained; partial success is more frequent; relapses are many, and failures not rare. Tic may develop in defective subjects; obviously where there is a gross mental deficiency, favorable results from any form of treatment can scarcely be expected.

CASES OF INFANTILE CEREBRAL PALSY PRESENTING CERTAIN PECULIARITIES

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The term "infantile cerebral palsy" has for some time been applied to a large and heterogeneous group in which many cases present hardly any marked "palsy," and in which many probably have lesions also involving portions of the central nervous system other than the pallium. The term, however, is not very inaccurate if it be taken to mean all cases of motor disturbances due to lesions of the cerebral hemispheres inclusive of the corpora striata. It is from twelve to twenty years ago that this group, or these groups, of cases were analyzed and systematized by Freud and other writers. Since then many cases have been regularly seen in routine clinical work, mainly for the purpose of ascertaining whether they fall in this general category and whether they offer any hope for improvement by surgical, orthopaedic or training procedures; but they are not usually studied intensively, clinically and anatomically. When this is done, light may be thrown upon the mechanisms forming the basis of voluntary movement, and possibly a better foundation laid for treatment.

It is not the purpose at present, however, to make any such ambitious attempt, but simply to put on record some cases in all probability falling within this group and presenting some peculiarities of interest.

CASE I. A. M. (23766), male, age four years. Date of first examination, Jan. 22, 1919.

HISTORY. *Present Illness.*—Complaint: He does not walk well. He does not talk at all. He does not understand as well as he should. He is very restless, does not feed himself, drools and is incontinent. He loses

breath and gets blue when he hurts himself.

PREVIOUS HISTORY. *Birth.*—A full term baby, easy labor, no instruments. Weight, seven and a quarter pounds. Asphyxia was present at birth—"he had to be slapped to make him breathe." He passed no water for five days. No indications of lues.

Development.—He was bottle-fed. First teeth at six months. He has never been able to get up to a standing position unassisted. He was able when assisted to stand, to walk a little alone at three and one-half years. He has never talked. He doesn't feed himself.

Diseases, Injuries, etc.—There is no history of injuries. He apparently never had convulsions, i.e., he would hold his breath and get blue, but there were no convulsive movements. He has had pneumonia five times.

Family History.—The mother's first baby was the patient, her second died at 18 days of "erysipelas of the intestines after circumcision," her third was still-born at nine months. A Wassermann test was made of the mother before this last birth, and she was told it was all right. The father also is living and healthy, excepting his "stomach is bad."

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—None were noted, except that when lying on his back the patient showed a tendency to draw up his legs in the manner characteristic of infants less than a year old.

Gait.—He walks unsteadily, with his feet far apart; he lifts his feet too high and stamps; in brief, his gait is infantile. He cannot rise to the standing position

without taking hold of something. He stands very unsteadily.

Coördination.—The coöperation was too poor to permit of definite tests, but his various acts were performed in an infantile manner.

Skilled Acts.—There was no speech; there were only infantile sounds.

Abnormal Involuntary Movements.—None were observed, unless the poorly coördinated irregular movements of infancy be characterized as choreo-athetotic.

REFLEXES. Deep.—The biceps, triceps and patellar are active. There is no clonus.

Superficial.—The abdominals are active. There is no Babinski.

Muscle Strength.—This could not be accurately tested on account of lack of coöperation, but appeared to be normal judging by the vigor of various spontaneous movements.*

Muscle Status.—No particular tests were made, but nothing abnormal was noticed.

GENERAL SENSORY.—This was not tested, owing to lack of coöperation and time, but nothing to indicate abnormal conditions was noticed.

CRANIAL NERVES.—The pupils react to light. The facial and tongue movements are normal, as far as could be judged by spontaneous movements.

MENTAL STATUS.—The child is evidently mentally defective, but understands apparently rather better than it reacts.

CASE II. A. C. (23760), female, age 15 years. Date of first examination, Jan. 22, 1919.

HISTORY. Present Illness.—The complaint is that she is defective mentally, that she doesn't know how to play or get along with friends, and that she is clumsy

in everything she does. She cannot comb her hair. When she carries anything she is liable to shake and drop or spill it. She is even clumsy in walking. She twitches very much. "She can sit and sit and think and suddenly begin to smile."

From infancy she was weak. She used to fall a great deal even up to ten or eleven years of age, and even now may fall now and then. After two years the doctor called attention to a weakness of her feet. She was taken to a hospital; nothing was found to be the matter with her feet, but a number of doctors agreed that she had chorea. At four or five years she was very nervous and easily frightened.

Her clumsiness is better now, but mentally she seems to her family to be unchanged.

PREVIOUS HISTORY. Birth.—She was a full term baby; easy delivery without instruments. Weight five pounds. No asphyxia. No indications of lues.

Development.—The first teeth came at four and one-half months. She began to walk at twenty months and to talk at two years. She began school at six years of age, but was a very poor scholar. She reached grade 3B at twelve, then left school for three years and went back last fall. She is now in the ungraded class. Menstruation began when she was twelve and a half.

General Physiological Status.—Her sleep is restless and appetite poor. Menstruation is regular, lasting 8 or 9 days, amount large and sometimes with pain. She has occasional diurnal incontinence of urine.

Diseases, Injuries, etc.—There is no history of important injuries, or of operations. She sometimes has slight headaches and complains of pains in hands, feet or body, the location being variable. There is no history of vomiting, but she sometimes regurgitates when she eats ("water brash"?).

Family History.—The mother has had no miscarriages or stillbirths. There are three children living beside the patient.

* This raises the interesting question as to whether the strength is normal, judged by force of voluntary effort of an infant of the age at which such movements are usually made, or judged by a four year standard. This could not be easily determined, but it would seem more likely to approximate the former than the latter.

They are healthy. One child died at one and one-half years of age (convulsions with whooping cough and pneumonia). The patient is the youngest child. The mother is healthy but was very sick while pregnant with the patient. She was in bed four months, vomiting and "water from the mouth." The father is living and healthy.

VOLUNTARY MOTOR SYSTEM. *Gait.*—The gait is rather awkward and unsteady—quite unsteady when walking on a straight line.

Coördination.—This appears to be normal with the exception of occasional choreiform movements.

Skilled Acts.—Speech appears to be normal.

Abnormal Involuntary Movements.—There are present occasional choreiform movements when performing various acts, as above noted.

Reflexes.—The deep reflexes (biceps, triceps, radial, suprapatellar, patellar and Achilles) are all active and equal. There is no clonus, and no Babinski.

Muscle Strength.—This is good in all parts (head, fingers, forearms, arms, feet, legs and thighs).

Muscle Status.—Nothing abnormal was noted.

GENERAL SENSORY.—Touch is probably normal. The patient was afraid of the cotton. Vibratory was not done, the patient showing fear after the first test. Muscle-tendon sense is normal in feet, legs and upper extremities.

CRANIAL NERVES. *Eye.*—The patient shows hyperopic astigmatism. The fundi are normal, and so are reactions to light. The right pupil seems slightly smaller than the left. The eye movements show no limitations, but are unsteady.

Face.—The facial movements are normal. There is some facial asymmetry.

Tongue.—The position and movements of the tongue are normal.

MENTAL STATUS.—The following condensed report was furnished by Miss E. Keller: The girl is chronologically in adolescence (15 years), but mentally she is about nine years of age.

Her attention and discrimination are good in concrete situations; but attention, concentration and memory (where words, not so much numbers, are involved) are poor.

Her chorea has prevented motor development.

She lacks confidence, and has some fears which interfere with her orientation and mental development.

If taught alone she could make some progress. She gets little in a group.

CASE III. R. G. (23738), female, age 13 years. Date of examination, Jan. 21, 1919.

HISTORY. *Present Illness.*—The patient had come to the clinic some three years previously and was tested mentally at that time. On the present occasion she came with a minor complaint, apparently, of pains in the back and side and occasional left frontal headache.

She began to have convulsions when nine months old. Sometimes she would have only one a year, sometimes one after another. The last one, described below, was the most severe of all.

Five years ago, early one morning, the mother found the patient in bed shaking and unconscious. "She shook first on one side and then on the other." Her teeth were tightly closed and she was blue. She bit her tongue and frothed at the mouth. The attack lasted four or five hours. She was weak afterwards. When she wanted to go to the toilet her mother noticed that she was crooked. She could not walk for two months. The right leg, arm and face were affected, the latter being drawn to the left. Previous to this she had been right-handed. She has had no attack since.

She has always been dull; before the last attack she had been "wild," but now she is "too quiet."

PREVIOUS HISTORY. *Birth.*—She was a full term baby with a normal delivery without instruments. Weight, six and one-half pounds. No asphyxia. No indications of lues. "She was a beautiful baby till about seven years of age when she had the stroke."

Development.—She was breast-fed. The first teeth appeared at two years. She began to sit up at nine months, to stand at one year, to walk at one and one-half and to talk at four years. She began school when six years old and is at present in the ungraded class. Menstruation began at 12 years.

General Physiological Status.—She sleeps well, appetite is good, bowels are regular. Menstruation is regular, every four weeks with a duration of one or two days, amount scanty, no pain. There are no bladder disturbances. "She was always clean."

Diseases, Injuries, etc.—There is no history of injuries. A tonsilectomy was recently performed. There is no complaint as to vision or hearing. No motor disturbances are present other than those above mentioned.

Family History.—This presents points of interest. The first child is now a girl of 19 years, is in good health, and graduated from High School with good marks. This child had convulsions from nine months to six years of age. The second child is a boy of 17 years, about to finish High School, and in good health. He had convulsions from nine months until he was 11 years of age. The third child is a girl of 15 years, in good health. She is bright, and has graduated from Grammar School. She had one convulsion at 11 months. The fourth child is the patient. After her there were three miscarriages, about ten weeks, apparently induced.

The mother is in good health. The father is also in good health. He had convulsions when about three years of age.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—There is a

slight pes cavus, more marked on the right foot. Both feet, when the patient is recumbent, tend to adduct, with the big toes dorsiflexed. This is more marked on the right than on the left. When the patient extends her arms, hands and fingers, the positions assumed by the two hands are different: the left fingers, hand and forearm are more in a straight line; the right hand is more volar-flexed at the wrist and the right fingers more dorsiflexed at the knuckles.

Gait.—The gait is nearly normal, there being a slight limp. The shoes are worn evenly.

Coördination.—Standing with the feet together, eyes open and shut, is normal. The patient can stand unsteadily on the left foot with the eyes open and shut, but cannot stand on the right.

The finger to nose test is performed normally with the left hand, but unsteadily with the right. The way this is done produces an appearance of ataxia but it seems more probable that it is due to the slight choreo-athetosis, or chorea, mentioned below. Tests with the lower extremities appear to show in a less degree the same phenomena on the right.

Pronation-supination and flexion-extension tests of the arm show an adiadochokinesis on the right. There does not appear to be a true dysmetria on the right.

Skilled Acts.—As already stated, the patient was right-handed before the paralysis. She is now left-handed and has learned to write, slowly but fairly well, with her left hand.

Speech appears to be nearly normal, perhaps rather drawing and measured at times.

Abnormal Involuntary Movements.—The right hand, extended, shows an unsteadiness of the fingers and hand, more choreiform than athetotic in character.

REFLEXES. *Deep.*—These are all active, but not very active. The biceps and triceps are more marked on the right; whether there is a similar difference in the lower

extremities is rather doubtful. At times the right patellar appeared more marked than the left. The uncrossed periosteal (tibio-adductor) is greater on the right. There are no crossed periosteals.

Superficial.—The abdominals are all slight on both sides, but are more easily elicited on the left. There is a slight Babinski on the right, a doubtful one on the left. No Chaddock, Oppenheim, Gordon or Schaefer.

Muscle Strength.—The right fingers are weaker than the left in flexion, extension, abduction and adduction, especially the last three movements. Flexion and extension of the right hand is weaker than the left.

Plantar flexion of the toes is much diminished on the right, dorsal extension is less diminished. Plantar flexion of the feet is good on both sides, dorsal flexion is less on the right than on the left. Abduction of both feet is weak, more so on the right. Beyond these points there was only noted a slight paresis in outward rotation and flexion of the right thigh.

In brief, the paresis on the right is most marked distally.

Muscle Status.—There is a general atrophy of the right hand and fingers.

GENERAL SENSORY. *Touch.*—Touch discrimination is normal or nearly so on the second finger tips, there being possibly a slight diminution on the right as compared with the left.

Pain.—This is normal on the fingers and toes (other parts not tested).

Muscle-tendon.—This is normal in the big toes and feet. There is possibly a slight diminution in the right hand.

Stereognosis.—Superficially considered, there appears to be quite a marked astereognosis. It was noticed, however, that it was most marked with small objects which the patient did not handle freely and that when these were placed between the finger tips they were better identified.

CRANIAL NERVES. *Eye.*—There is some blurring of the nasal halves of the discs,

more on the right; the vessels are large and there is slight temporal pallor, appearances not infrequent in this type of cases. The pupils are of normal size, equal, and react well to light and convergence. There is no strabismus or abnormality of the eye movements, and no abnormality of the palpebral fissures.

Trigeminus and Mouth.—Nothing abnormal was noted.

Facial.—The facial movements are normal. The right face is somewhat smaller than the left.

Glossopharyngeus, Vagus.—The position of the uvula is normal.

Hypoglossus.—The position and movements of the tongue and articulation are normal.

MENTAL STATUS.—The following condensed report was furnished by Miss Keller: This girl was examined in September, 1915, when she was chronologically nine years and three months old. Mentally her reactions were like a girl of about six in observation, orientation, memory, discrimination. In abstract work, as seen in number work, she was only about four years old.

On being reexamined, February 1919, when over 12 years old, she shows some mental improvement, possibly a year. Her number work, though slightly better, is only that of a six year old girl. She is poorly oriented in time, her memory for form is poor, but observation, discrimination and some reasoning in simple concrete situations show growth. She has had to learn to write and draw with the left hand, and has shown ambition and concentration enough to succeed. She seems to be conscious of her shortcomings, and attention is improved.

Volitionally and emotionally she is normal.

SYSTEMIC.—The heart is normal.

CASE IV. F. W. (23672), male, age eight years. Date of examination, Jan. 15, 1919.

HISTORY. Present Illness.—The complaint consisted of the following symptoms:

He doesn't do well in school, he can't seem to remember. He is babyish. He has always been so.

There is something wrong with his walk. He seems to give way at the knees on both sides. He doesn't fall. The mother seems to have noticed this only during the past half year or so. He has always had trouble with his ankles; they are weak, especially on the right.

He is awkward in handling things, spills, etc. He has always been so.

His shoulders pull and pull all the time. This has been noticed only for about a year.

PREVIOUS HISTORY. Birth.—He was a full term baby (ten months according to the mother), a prolonged delivery with instruments. Weight about eight pounds. There were cyanosis and asphyxia.

Development.—The first teeth appeared at 13 months. The fontanelles closed later than his brother's. He began to walk at 23 months, and to talk at three years, then only one word at a time. His younger brother (now five and a half years old) talked first. He began school at six and a half years, and is still in grade 1. It is proposed to put him in the ungraded class.

General Physiological Status.—He sleeps well, his appetite is good and his bowels are regular. He has diurnal incontinence, but no nocturnal enuresis.

Diseases, Injuries, etc.—There is no history of injuries, convulsions, pains, headaches (except one three weeks previously) or vomiting. He had measles at seven years.

Family History.—The patient was the mother's first pregnancy. The second was a boy now five and a half years old, healthy and bright. After this there were two miscarriages of about six or seven weeks attributed by the mother to coughing due to an asthma she had contracted previously to these last two pregnancies.

The mother is healthy except the asthma she has had for the past four years. The father is in good health.

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—There is a tendency to pes cavus, more marked on the left. There is pes planus on the left when standing.

Gait.—He is rather unsteady in gait, and seems possibly especially awkward with the right foot. The sole of the right shoe is worn badly on the inner side.

Coördination.—He shows slight unsteadiness and a few jerky movements, both in standing and in performing the various tests. *Adiadochokinesis* is present, but it is doubtful whether there is any *dysmetria*.

Abnormal Involuntary Movements.—The arms and hands, when extended, show some choreiform movements. The movements of the fingers are rather slow but not *athetotic*.

REFLEXES. Deep.—The biceps, triceps and radial are active, the suprapatellar and patellar are very active, the Achilles active. There is no clonus.

Superficial.—The abdominal reflexes are rather slight. There is a doubtful Babinski on the left, also apparently an Oppenheim and Gordon, but no Chaddock or Schaefer. When the foot was held at right angles to the leg the Oppenheim was not so constant.

Muscle Strength.—The coöperation was poor, but there seems to be some weakness in hands, fingers, thighs, legs and feet.

Muscle Status.—Certain phalanges of the fingers and the legs at the knees are hyperextensible.

GENERAL SENSORY. Touch.—This is normal as far as can be ascertained. Pain is normal. Vibratory present in big toes, ankles, shins, elbows and knuckles. Muscle-tendon sense appears to be poor in big toes and feet but the coöperation was probably not good. Imitated movements of the upper extremities are good.

CRANIAL NERVES. Optic.—The pupils react well to light. The eye movements

are normal excepting a slight transient nystagmus to the left, scarcely of significance.

Facial.—This is normal. No abnormal involuntary movements were noted. There is a tendency to drool.

Hypoglossus.—The position and movements of the tongue are normal, except that it is unsteady when protruded.

MENTAL STATUS.—The boy is mentally defective.

CASE V. H. C. (23965), male, age 11 years. Date of examination, Feb. 8, 1919.

This boy has attended the clinic before and was briefly examined April 27, 1916 (7180). The following were the observations then recorded: He was sent home from school because he was not quiet enough. He holds his head retracted and twists and turns it while in this position. He does not wish to be undressed, striking and kicking his father while he is doing it. Reflexes are active but equal.

HISTORY. Present Illness.—The boy looks rather dull and apathetic. He smiles at pleasantries. The complaint (by the father) is that he "shivers" and "don't speak loud and so they keep him in the ungraded class." He has always been so and has always spoken indistinctly. By shivering, the father means that when he wants to write his hand shakes.

On the third day after birth he lay unconscious for half a day and his face and eyes became yellow. The father attributed this to the spell of the evil eye exercised by a woman who was in the room soon after his birth. He was taken to Beth Israel Hospital (dispensary) two or three days and then seemed all right.

PREVIOUS HISTORY. Birth.—He was a full term baby, not a prolonged labor, and no instruments. Weight over 10 pounds. There was no asphyxia, and no indication of lues.

Development.—He was breast-fed, his

first teeth came "late." He began to walk and talk at two years. There was nocturnal enuresis until he was five years old.

Diseases, Injuries, etc.—There is no history of injuries. No convulsions. No special complaint of pains or headaches. No vomiting. He had measles at four.

Family History.—The mother has six children living and healthy and bright, except the patient. None have had convulsions. There were no miscarriages or stillbirths. One child died in 1916 when 18 months old of paralysis (infantile, no convulsions). The father and mother are living and in good health. The father had a blood test from the arm eight years ago, he says, taken at a hospital and sent to the Board of Health. He was told it was all right.

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—There is a slight kyphosis and tendency to winged scapulæ and a "round-shouldered" effect, especially on the left. The neck is thick, with an appearance as though the head had been pressed downward. He always tends to stand with one leg flexed. There is pes planus.

Gait.—The gait is rather irregular.

Coördination.—Standing and performing the various tests, coördination is normal except for choreiform movements.

Skilled Acts.—The speech was not tested, but the patient speaks very little, in a low voice and with rather defective speech.

Abnormal Involuntary Movements.—There are choreiform movements of all parts, including eyes, face, tongue and phonation apparatus.

REFLEXES. Deep.—The biceps, triceps and radial are active. There is transient clonus of the suprapatellar, patellar and Achilles. There are no periosteal (tibio-adductor) or crossed patello-adductor reflexes.

Superficial.—The abdominals are active. There is no Babinski or any of its modifications.

Muscle Strength.—The patient could not rise from the recumbent to sitting position at first, but this was not due to weakness, he being soon taught to do it. There appears to be no paresis in fingers, hands, forearms, arms, elevation of shoulders, thighs, legs, feet and toes, except an inability to abduct the right foot and to flex the toes well.

Muscle Status.—There appears to be more tonus in the lower than upper extremities.

GENERAL SENSORY. *Touch.*—Hastily tested, normal. Pain normal. Vibratory is normal in big toes, shins and elbows. Muscle-tendon is normal in big toes, feet and upper extremities. Stereognosis is good in the right hand, the only one tested.

CRANIAL NERVES. *Optic.*—The fundi are normal. The pupils react well to light. The eye movements are normal except unsteadiness due to chorea.

Facial.—The facial movements are normal except choreiform movements.

Glossopharyngeus and Vagus.—The tip of the uvula deviates to the right. The palatal movements are normal.

Hypoglossus.—The position and movements of the tongue are normal except that he cannot elevate it. He can depress it and move it laterally.

MENTAL STATUS.—The following condensed report was furnished by Miss Louise E. Poull, Department of Ungraded Class: 1-31-1919. Borough Bk. Public School 43.

Chronological age ten years ten months. Mental age six years eight months. Intelligence quotient 61+ (Stanford Revision of Binet tests). A mental defective of nervous type.* Development of powers unusually even. Attitude is in accordance with his mental age. Alternative tests were used when motor difficulty invalidated a test. Shows interest in the work of the

tests and does not attempt things too difficult for him. Shows no outstanding ability. Can follow manual directions. Social comprehension of about 6 years. Recommendation: To remain in ungraded class.

SYSTEMIC.—The heart is normal.

CASE VI. B. S. (23309), male, age seven years. Date of first examination, Dec. 9, 1918. Examined and diagnosed by Dr. John Nolan.

HISTORY. *Present Illness.*—The complaint is restlessness and twitching of the fingers. The child is now extremely restless. When he eats his hand shakes. He could not write in a straight line. He is tonguetied in his speech. His head constantly shakes without any formula and without any jerking or sharp twitching. Soup is always spilled when he tries to carry the spoon to his mouth. This condition has always been present. He is better than formerly.

PREVIOUS HISTORY. *Birth.*—He was a full term baby, seven hours labor, no instruments. Weight seven pounds. No cyanosis. No keratitis, scars or other signs of lues.

Development.—He was bottle-fed. The first teeth appeared at seven months. He began to hold up his head at four to five months, to sit up at one year, to stand at 14 months, to walk and talk at 18 months. He began school at five years, but his school career was interfered with by his motor disturbances.

General Physiological Status.—Nothing abnormal noted. There is no incontinence or nocturnal enuresis.

Diseases, Injuries, etc.—There is no history of injuries. The tonsils were removed at six years. There is no complaint of pains or headaches. He had pneumonia at four years, measles at three, croup after measles twice.

Family History.—The mother had no

* There seems here to be a confusion between nervousness and chorea.

miscarriages. There was one stillbirth, eight months. "The doctor said the heart didn't close." The first pregnancy was a girl now 18 years, in good health. The second was a girl, 14 years old, now in Vineland. She cannot walk well, twitches and cannot talk. She is said to be feeble-minded. The third, a boy of ten years, is in good health. The fourth is the patient.

VOLUNTARY MOTOR SYSTEM.—Attitude and gait are normal except some choreiform movements.

Coördination.—Equilibratory and non-equilibratory are normal, except choreiform movements.

Skilled Acts.—He has a choreiform dysarthria.

Abnormal Involuntary Movements.—Choreiform movements are present in hands, feet and tongue.

REFLEXES. *Deep.*—The biceps, triceps and radial are all active. The suprapatellars are active, the patellars very active, the Achilles active.

Superficial.—The abdominals and cremasteric are rather slight. There is no Babinski or any of its modifications.

Muscle Strength.—This is normal in movements of the head, fingers, hands, forearms, arms, thighs, legs and feet, except as interfered with by the choreiform movements.

Muscle Status.—There are no atrophies. The muscles are rather hard and there is some hypertonus.

GENERAL SENSORY. *Touch.*—Touch acuity is normal on all parts (hastily tested). Touch discrimination of the second finger tips and big toes was tested and found to be normal. Vibratory was present and probably normal on ankles, shins and ulnars. Muscle-tendon was normal in big toes, feet and upper extremities.

CRANIAL NERVES. *Optic.*—The fundi are normal. The pupils are 4 mm., equal, circular and central. They react well to

light and convergence. There is no strabismus, exophthalmus or enophthalmus. No nystagmus.

Facial.—The facial movements are normal except for choreiform movements.

Glossopharyngeus and Vagus.—Normal.

Hypoglossus.—The position and movements of the tongue are normal, except for a choreiform unsteadiness.

MENTAL STATUS.—The following condensed report was furnished by Miss E. Keller: Chronologically seven years old, this boy is mentally the same age. His power of concentration and attention is affected by the restless habits due to chorea. His ability to perform tests of doing is also outwardly unsatisfactory, but in execution he reasons and judges. His discriminative ability is a little below par.

SYSTEMIC. *Tegumentary.*—Normal.

Glandular.—Normal.

Cardio-vascular.—Pulse is normal.

Respiratory, gastro-intestinal, genito-urinary and skeletal systems are normal.

CASE VII. B. H. (23364), female, age 11 years. Date of first examination, Dec. 14, 1918.

HISTORY. *Present Illness.*—The complaint is stiffness of the right arm and leg and mental backwardness. When two weeks old she had inflamed eyes because "they were not properly cleaned at birth" and they were treated with a wash every 15 minutes. "This weakened her and the milk did not agree with her."

When three months old she had convulsions lasting two weeks, at the worst with intervals of one or two hours. She tightened her hands, body and legs, and her eyes rolled sideways. Twice she seemed as though dead.

When two years old paralysis was first noticed. She couldn't stand or walk. The right leg was shorter than the left. The right fist was clenched and the right arm flexed.

PREVIOUS HISTORY. *Birth.*—She was a full term baby, the labor was not prolonged and no instruments were used. There was no asphyxia.

Development.—She began to walk at five years, to talk (?).

Diseases, Injuries, etc.—There is no history of injuries. A cord in the leg was cut when she was four years old. There is no complaint of pains or headaches. She has had measles, influenza and pertussis, and is subject to boils.

Family History.—There were no miscarriages or stillbirths. One child died at six months of summer complaint. There are five living children, all healthy except the patient. The father is living and healthy; the mother is also living and healthy but "nervous."

VOLUNTARY MOTOR SYSTEM. *Gait.*—She walks with the right leg stiff at the knee, the right foot somewhat rotated out and abducted, the toes sometimes plantar flexed and sometimes all turned outwards. She cannot walk on tiptoe. The toe of the right shoe is worn. She usually wears a smaller shoe on that foot.

Coordination.—She is left-handed. Coordination is normal except the paralytic disability.

Abnormal Involuntary Movements.—There are slow athetotic movements of the right hand and foot, more marked in the hand.

REFLEXES. *Deep.*—The biceps, triceps and radials are rather slight and are equal, except possibly the latter. The suprapatellars, patellars and Achilles are active and equal or possibly slightly greater on the right. There are no periosteals.

Superficial.—The abdominals are all slight but apparently equal. There is no Babinski or any of its modifications.

Muscle Strength.—Rising from recumbent to sitting position is normal. There is paresis in abduction and adduction of the right thigh, possibly in flexion and extension of the right foot. Flexion and

extension of the legs are normal. Athetotic remissions in resistance to passive movements were noted on the right. Extension of the thighs seems good. She cannot hold the right fingers extended and *adducted* or adduct the right hand. Strength here is good otherwise. Flexion and extension of the arms are good; rotation, both supination and pronation, weak.

Muscle Status.—There is an atrophy of the right leg and foot. The right leg is harder than the left.

Abnormal Associated Movements.—While drawing up the legs against resistance she dorsiflexes the left foot and plantar-flexes the right foot, sometimes adducting the right foot. There is a tendency to pronation and palmar-flexion of the right arm and hand.

GENERAL SENSORY. *Touch.*—Acuity is normal. Discrimination is good on the second finger tips. Vibratory is normal. Muscle-tendon is probably normal. Stereognosis is fair.

CRANIAL NERVES. *Optic.*—The fundi are normal. The pupils react well to light. There is a concomitant convergent strabismus with hyperopia. No nystagmus.

Facial.—She has difficulty in holding her eyes shut. There is an asymmetry of the face, but the lower facial movements are normal.

Hypoglossus.—The position and movements of the tongue are normal.

MENTAL STATUS.—The following condensed report was furnished by Miss Louise E. Poull: 12-12-1918. Borough M. Public School 132. Age 11 years two months. Mental age six years six months. Intelligence quotient 58+ (Stanford Revision of Binet tests). Mental defective of middle grade. The basal age is six years and balance of powers is fairly equal. Performance tests are probably difficult for her on account of neurological defect. She is left-handed. Progress in ungraded class is slow.

CASE VIII. F. P. (23325), male, age eight years. Date of first examination, Dec. 11, 1918. Examined by Drs. Oliver S. Strong and John Nolan.

HISTORY. Present Illness.—The complaint is that he is very nervous and that he is backward. He has always been so. His joints were very loose up to six years of age. He tired easily. He complains now of getting tired easily, nevertheless he runs about all day. "His legs hurt," he says. He plays with boys of the same age. He does not quarrel; is afraid. He cries easily and is excitable.

He complains of headache about once a week. This is relieved by catharsis and does not last long. There is no vomiting.

PREVIOUS HISTORY. Birth.—He was a full term baby; the labor was prolonged and instruments were used. There was no asphyxia. No indications of lues. He was born with a supernumerary, ectopic eyelash, consisting of a small tuft of hairs on the right upper lid.

Development.—He was breast-fed. The first teeth appeared at eight months. He began to walk and talk at twenty months. He entered kindergarten at five years and regular school at seven. He reached 1B and was transferred to ungraded class for observation.

General Physiological Status, etc.—He sleeps and eats well. There is no incontinence and no nocturnal enuresis.

Diseases, Injuries, etc.—There is no history of injuries. Adenoids and tonsils were removed at six years. There were no convulsions. He sometimes vomits when terrified, as, for example, by the sight of blood. He had chicken-pox last January and measles last May.

Family History.—There were no miscarriages or stillbirths. The mother's first pregnancy was the patient; the second, a girl of five years, healthy and bright, without motor disturbances; the third, a child of three years also bright and without motor disturbances. The girl has

some hyperextensibility of arms and fingers and slight reflexes. The parents are living and in good health.

VOLUNTARY MOTOR SYSTEM (coöperation poor).—Coördination was found to be good as far as could be tested. No abnormal involuntary movements were noted outside of a general restlessness.

Reflexes.—No deep reflexes can be elicited, and the abdominals only with difficulty. There is a doubtful Babinski on the left; no modifications of the Babinski are present.

Muscle Strength.—Poor coöperation prevented careful testing. There is apparently some weakness of the upper extremities.

Muscle Status.—There is hyperextensibility at the elbows, less elsewhere.

GENERAL SENSORY.—This could not be well tested. Pain is present and muscle-tendon sense was found to be present in the big toes.

CRANIAL NERVES. Optic.—The fundi are normal. The pupils react well to light. The eyes are quite prominent. The eye movements are normal.

Facial.—The facial movements are normal but there is poor control.

MENTAL STATUS.—The following condensed report was furnished by Miss E. Keller: This child is chronologically seven and a half years, mentally about five years old. His powers of discrimination and concentration are poor. He is eye-minded, and through visual memory takes in number and form and retains it. He shows no judgment or reason in simple concrete problems. Recommended that he stay for the present in 1A or B if the school is willing to retain him.

The following condensed report was furnished by Miss Louise E. Poull: 12-5-18. Borough Bk. Public School 123. Age eight years five months. 1B grade. Mental age four years eight months (Stanford Revision of Binet tests). Intelligence quotient 55+. Mental test shows marked

asymmetry of function. Power of concentration is probably not above the fourth year. Attention is distractible. He shows poor discrimination of form, but is able to follow simple directions in manual work. Practical social comprehension and orientation are about the fifth year. School History: Has been two terms in 1A and attainment at present is not above that grade. Cries frequently but is not vicious. Attendance has been irregular on account of sickness. A re-test is recommended if physical conditions can be improved.

LABORATORY TESTS. — Blood Wassermann negative. Spinal fluid. Cells 0; globulin, a trace; Wassermann 0 to 2.0; colloidal gold 0 0 0 0 0 0 0 0 0 0.

All of the preceding cases, in the opinion of the writer, have some defect of the cerebral hemispheres, including the basal ganglia; and it is doubtful if any of them have defects elsewhere in the central nervous system, though these cannot be certainly excluded. These defects, however, with our present imperfect knowledge of cerebral mechanisms of movement—and mentality—cannot be accurately localized, and the writer will simply point out what appear to him to be points of especial interest.

CASE I presents a lack of development of the mechanisms for skilled acts, including such fundamental acts as standing and walking. These, as far as they can be performed, are done in a way apparently indistinguishable from an infant who is learning to stand and walk. There appears to be no outstanding single defect of any kind, and probably the best characterization of the picture would be to call it a dyspraxia, not, however, in the sense of a disturbance of a praxis already acquired. The uncertainty of standing and gait present a picture rather more like an ataxia; but while such a point can hardly be settled by observation, it seems more probable that there is no real lack of sensation, including deep sensation, but

more of the associative cerebral mechanisms which build them into definitely acquired acts.

CASE II presents a mental defect which is, perhaps, of no special interest in itself except as showing that the pallium is involved. Besides this, however, there are an unsteadiness of gait, not very marked, and a general awkwardness in the performance of other acts. Although the patient is mentally defective she is nevertheless mentally nine years of age, an age long past that at which such acts should be perfectly performed. The dysbasia and dyspraxia, if we can call them such, are then outstanding features and not simply a part of the general mental defect. In accounting for this an important factor must be taken into account, the presence of chorea. There is a history of chorea and there are at present apparently some remnants of this trouble. The slight chorea present now does not seem to be a cause of the dysbasia, etc., but the question arises whether a more severe and protracted chorea earlier in life might not have interfered with the child's acquisition of an eupraxis. In a true dyspraxia, the child's associative cerebral apparatus would be defective. A chorea, however, is probably a disturbance of principal or accessory efferent paths, or both, preventing the performance. This would not materially affect the praxis mechanism if occurring in an individual who had already acquired an eupraxis, but might if occurring at an early age before this acquisition had taken place. The impression one gets from the patient is rather against the chorea accounting here for the awkwardness; and this explanation is further invalidated by the findings in Case VI, where permanent chorea does not seem to have produced such an effect.

CASE III.—This case presents points of etiological interest also, i.e., the history of repeated convulsive seizures terminating in a hemiplegia, since which the attacks have ceased and the patient seems changed in disposition. There is also the interesting

history of convulsions in other members of the family. The remnants of the hemiplegia are still seen in the alterations of the reflexes already described, in certain pareses, and atrophy, involving especially the distal portions of the right hand and foot, and certain other peculiarities of these two members. It seems probable that the mental defect, convulsions and hemiplegia, are all due to the same pathologic process.

The peculiarities shown by the right hand, the apparent astereognosis and particularly the interpretation of the movements it exhibits while performing coordination tests appear especially interesting. These movements present quite an ataxic appearance. When the hand and fingers, however, are extended, they show a movement which may perhaps be characterized as choreo-athetotic, though they are more choreiform than athetotic in character. These movements are not very marked, and the question arises whether there is any true ataxia. If there is, we would expect to find in this case a defect in muscle-tendon sense. There were some indications of a slight defect here, but the case was hardly examined often enough to make sure of this point; and it must be remembered that a hand that is used little may show a slight apparent loss in this respect. The question as to the presence of a true astereognosis is, of course, closely associated with the above. The loss in ability to identify objects placed in the right hand was quite marked, but the patient did not handle the objects well, especially with the fingers, and identified them better when they were placed between the finger tips. Larger objects, not requiring so much manipulation with the fingers for identification, were identified better. Touch discrimination was normal or nearly so. It would seem then that with a slight sensory defect, paresis, and a lack of use of the hand due to the above and to the abnormal involuntary movements, and resulting in practically a dyspraxic condition, there would hardly be any true astereognosis,

the apparent agnosia being explained by the above defects. The interpretation of such phenomena is admittedly difficult, and it is hoped that a further study can be made of this case.

Whether the lesion is cortical, or sub-cortical, or located lower in the forebrain, depends somewhat upon the view taken as to the locations of lesions producing abnormal involuntary movements, especially those of a choreiform character. The history of convulsions and mental defect proves, especially the latter of course, a pallial lesion. Whether the presence of the abnormal involuntary movements necessitates the postulation of a lower lesion as well is perhaps an open question.

CASE IV.—This case is rather similar to Case II. The suspicion was raised here of a possible early Friedreich's ataxia. The pes cavus and unsteadiness with some apparent choreiform movements are compatible with this theory, while the active deep reflexes, though rather against it, are not incompatible. The suspicion was raised partly by the mother's statement that the unsteadiness, or giving way at the knees, had only been noticed lately. Probably at present this possibility cannot be entirely excluded.

An interesting point has already been raised in the history of this case in connection with the apparent presence of the Oppenheim and Gordon signs. The pes cavus on the right, with the tendency to dorsiflexion of the great toe, seemed to give a predisposition, as it were, to such a reaction, which view was apparently confirmed by a diminished tendency for the Oppenheim to appear when the foot was held flat and at right angles to the leg. This raises the further question as to what value should be attached to the sign in such a case. It would still seem to have value, because it must be remembered that the pes cavus and associated dorsiflexion of the toe itself have much the same significance as the various Babinski reactions.

A similar question occurs with the Babinski so frequently elicited in athetosis. The athetotic movements of the toes often assume the typical Babinski position. Such athetotic movements are in turn easily elicited or increased by various stimuli, especially, as associated movements, by muscular effort in other parts, but also by cutaneous stimulation; yet, in some cases at least, a Babinski may fail to be elicited by plantar stimulation when care is taken not to start a general athetotic reaction. Does such an absence of Babinski tend to show that the pyramidal tract has not been involved? No one would probably take this position. The explanation of the Babinski sign is probably wrapped up in the larger question of the mechanism of spasticity, associated movements, athetosis and chorea. Two opposed and an intermediate view may be taken as to the two latter phenomena: they may be produced by a lesion of the pyramidal system, by a lesion of an extra-pyramidal system, or systems, or by both. The resemblance between choreo-athetosis and infantile movements which later are replaced by or modified into the more definite acts presumably principally carried out by the later developing pyramidal system, the close connection between choreo-athetosis, especially athetosis and upper motor neuron paralysis, and the greater liability for the postparalytic athetosis to appear in infantile paralyses where presumably the infantile movements have not been so completely replaced by the acquired movements,—all favor the first view, which would place the performance of these movements upon extra-pyramidal mechanisms. The movements in question are also allied to or a part of the emotional reactions and pain reactions of a more generalized, unsystematized and convulsive character than the skilled acts, speech, etc.; and in pseudobulbar palsy we sometimes see a dissociation between these two categories. The Babinski reaction would be a part of these reactions, and might, according to such a view, be a part

or a vestige of these extra-pyramidal reactions, and the same would apply to the abnormal associated movements.

The prevalent view, however, at present, is hardly the above view, but rather that athetosis, etc., is produced by *lesions* of the extra-pyramidal system or systems. This view is supported by the occurrence of choreo-athetosis in non-pyramidal lesions (e.g., the superior cerebellar peduncle), and by the disappearance of normal associated movements in paralysis agitans where the lesion certainly does not appear to be pyramidal. Those who would uphold this view, however, do not always seem to bear in mind that if rigidly adhered to it would place athetosis, etc., in the category of *pyramidal* movements, which would hardly be acceptable.

The third view, attributing the abnormal movements to a partial lesion of the pyramidal system or a partial lesion of both pyramidal and extra-pyramidal mechanisms, removes some of these objections as well as one objection to the first view not mentioned, i.e., that, according to it, the extra-pyramidal system would be functionless or play no part in the performance of a skilled act. It may be pointed out here that the spasticity seen in upper motor neuron paralysis involves the muscles not paralyzed or only incompletely paralyzed, not those completely paralyzed, according to Lewandowsky. A still greater diminution of paralysis—and spasticity—might give rise to an athetosis; in fact, the athetosis may be practically movements of unequally spastic parts. The great bulk, perhaps all, of the cases with athetosis, etc., appear in a general way to have lesions which might interfere with intra-cortical and cortico-thalamus-striate associations. The choreiform element is possibly furnished by the cerebellar component present in the forebrain, though in the latter case we are again confronted with the fact that apparently a *lesion* of a cerebellar connection produces choreiform movements. Only further careful clinical examinations, coupled with minute postmortem studies,

can clear up such problems; but there may be some advantage in making the problems involved clearer by previous discussions of a more theoretical nature.

CASES V and VI.—These cases resemble each other in having what would appear to be a congenital chorea; at least according to the anamnesis they have always had choreiform movements. The cerebral character of the lesion in V is shown by the mental defect, and its probable affinity with a Little's diplegia by the hypertonus (not marked) and by the increased deep reflexes of the lower extremities. The abnormal positions of the back and shoulders are not improbably associated with the neurological defects, and are similar (but less pronounced) to those exhibited by another case seen in the clinic recently where there was also the dislocation of the hips occurring occasionally in such cases. Possibly such a deformity may be to a certain degree produced by a partial atonic-astatic cerebral infantile palsy of Foerster.

In Case VI there was no mental defect and no deformity; but even here there was some hypertonus and hyperreflexia of the lower extremities.

The outstanding feature of the two cases is the chorea, and there can be but little doubt that we are dealing here, not with a chorea minor, but with a symptomatic or postparalytic (or paraparalytic?) chorea such as also occurs after infantile hemiplegias. Neither patient appears to have had a history of paralysis, hence the term paraparalytic suggested above. The chorea does not appear to have interfered with the development of a good praxis in case VI, if the interference by the choreiform movements be deducted from the picture.

It is interesting to note that Case VI has a sister of 14 years in Vineland. It is

stated that she cannot walk well and cannot talk, also that she is feeble-minded. One cannot help wondering if the latter statement is true and whether she may not have simply a severe chorea.

The frequency of chorea of this type in children, especially those who are mentally defective or belong to the cerebral infantile palsy group, appears to be underestimated by writers, sometimes not being mentioned among the various types of chorea. It would be of interest to observe a case in which this occurred very early in life, and to note whether the chorea was simply a retention of the infantile movements. It will be noted, however, that in Case I there is no very marked choreiform effect in his movements.

CASE VII.—This is quite a typical case of infantile hemiplegia with spasm and athetosis. As seems to be almost the rule in these "palsies," the palsy is not very great.

An interesting point in this case is the slight change in the reflexes, the deep reflexes of the upper extremities being slight on both sides and those of the lower not especially active, and there being no marked inequality. Besides this, there was no Babinski and none of its modifications.

CASE VIII.—The interesting feature of this case, aside from an interesting mental picture, is the almost complete areflexia, the deep reflexes being absent and the abdominal reflexes very slight. The boy's past history seems to show an atony, but at present this is not marked. One can hardly invoke Foerster's atonic-astatic cerebral palsy here because in the Foerster palsy the deep reflexes are intact in spite of the atony. It is rather difficult to account for this condition, though similar observations have been made before on cases falling within this general group.

THE RESULTS OF OPERATION IN A CASE OF MENINGO-ENCEPHALITIS

BY NORMAN SHARPE, M.D.

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Patient: S. D.; age four years; male. History, meningo-encephalitis with epilepsy. The patient is the second of three children; the other two are living and well. There were no miscarriages. Labor was not difficult. No instruments. The patient was well and an apparently normal child until he was one year old. At that time he walked and talked a little. When thirteen months old he had an illness with high fever and convulsions, and lay, at times, with retracted head and eyes drawn to one side. The number and duration of convulsions not remembered. The child has not talked since that illness. He walks now, but stumbles and falls often. When three and a half years old, he began to have convulsions, general in character, with loss of consciousness. Attacks now come on, four or five a day. He does not pay attention, is bad tempered and destructive. He was examined in a neurological clinic and pronounced an idiot, with mild hydrocephalus.

Examination made April 27, 1916, reported a fairly well developed child with rather large head, wild-eyed and tossing about, having to be restrained in bed, alternately laughing and screaming. At first glance, the diagnosis of idiocy seems justified, but closer examination shows that he can understand and will obey commands if his attention is secured. The reflexes are normal and equal. There is no spasticity and no Babinski. Aside from his behavior, there were no stigmata of idiocy found. The Wassermann was negative. Fundi—discs were of normal appearance but veins were large and distended. The head was of mild external hydrocephalic type. Lumbar puncture showed fluid under pressure. It was decided to operate, with the view of relieving this increased and abnormal pressure.

OPERATION MAY 1, 1916

Right sub-temporal decompression. An area of bone $2\frac{1}{2}$ " x 2" removed under the temporal muscle on right side. Dura very thick and vascular. On incising dura fluid spurted 2", and there was much subdural fluid present, though not sufficient to justify a diagnosis of external hydrocephalus. The arachnoid was thickened and slightly grayish around the vessels.

Post-operative Notes.—Day after operation child is quiet and his attention is easily secured. One week later, child much quieter, out of bed, feeds self and tries to talk.

Child discharged in two weeks. Mother says he is quieter and better behaved than for years.

Bulging of wound present and decompression on opposite side advised later.

Three weeks later. Thirteen attacks since operation, but they are milder, involving only the head with no jerking of limbs.

In the following month had no attacks. Is much better and quieter than before operation, but still far from normal. Operation wound still bulges and second operation advised.

SECOND OPERATION JUNE 15, 1916.

Left sub-temporal decompression. Much fluid found under pressure. Appearance and condition of cortex same as at previous operation.

One month later, child has begun to talk, is much better behaved; has had no attacks since operation, and is not as nervous and timid as before.

Six months later, he had one general convulsion, the first since the second operation. He is talking better, feeds himself, understands and obeys commands, but is wild and unruly at times.

During the following year he steadily improved in behavior and mental development, though noisy and restless at times. The attacks returned in the early part of 1917 and continued throughout the year, mild in character and occurring about once a week. He disappeared from observation and was not seen until January 8, 1919, when he came to the clinic. He was so much changed for the better that he seemed another person. His restlessness was only slight, he was self-conscious, talked readily, could count, and identify money, etc. He is now nearly seven years old, and has had no convulsions for the past ten months.

The most important point in this case, second only to the remarkable improvement following operation, is that it was a condition usually considered not amenable to operation—meningo-encephalitis. In spastic birth palsy, due to hemorrhage in the brain cortex at labor, in a certain proportion of the cases there is an inflammatory reaction in the meninges with consequent retention of fluid and an abnormal increase of pressure within the cranium which may persist for years and permanently impair the child's physical and mental development. Whether this abnormal pressure is present or not, is readily ascertainable by ophthalmoscopic examination of the fundus of the eye and by the use of the spinal mercurial manometer at lumbar puncture. In those cases in which increased pressure is found (between 20 and 30 per cent of all cases of birth injury) it has been well established in the last few years that a decompression operation will be followed by definite and at times remarkable physical and mental improvement. But the child just reported was definitely not a case of birth injury. He was normal during the first year, had begun to walk and talk at 12 months, and at the 13th month there is a definite and clear history of "brain fever," with retracted head and convulsions—a meningo-

encephalitis. The patient did not walk again until three years old, and then badly, and did not regain speech until after operation.

As stated above, from the pathology of the condition, we have not considered meningo-encephalitis susceptible of benefit by operation. This was due to the fact that in a rather extensive series of cases examined, we found increased intracranial pressure in but comparatively few instances, and in some of those the mental impairment was so extreme that operation was not considered. Naturally, had not evidences of abnormal intracranial pressure been found in this case, operation would not have been done. If signs of increased pressure are lacking, cranial decompression is useless and will do no good.

While it is unwise to attempt to deduce too much from a single case, this one should serve to stimulate doubt as to whether we have not been too prone to consider all cases of meningo-encephalitis irremediable. Though I can give no approximate percentage of these cases that might be benefited by operation, I would consider it from observation to be far smaller than that of spastic birth palsies due to hemorrhage which, as stated above, is between 20 and 30 per cent of all cases of cranial birth injuries.

The two facts in this case to be emphasized are:

1. That it is unwise to diagnose idiocy without a complete and thorough use of all available methods of examination.
2. That in certain selected cases of meningo-encephalitis showing definite signs of increased intracranial pressure, operation may be followed by definite and even remarkable improvement. In this case the improvement indicates that the disease process had attacked chiefly the membranes, and had only slightly affected the nerve tissues; and the symptoms were largely due to the increase in intracranial pressure.

SCLERODERMA AND ALLIED TROPHIC CONDITIONS

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The study of what might, for want of a better term, be called the mechanism of the groups of symptom-complexes classified under the terms trophic and vasomotor neurosis, presents a problem for determination. These manifestations are not receiving the attention they deserve, partly perhaps because of their complexity, and partly by reason of the fact that their manifestations are often associated with diseases in which the vasomotor and trophic symptoms are but a part. As a matter of fact, trophoneuroses and vasomotor neuroses are probably not entities at all, and the clinical elements might best be studied as they appear in the course of the various lesions of the central, peripheral and autonomic nervous system and of the endocrine organization.

There is such a striking similarity in the clinical manifestations of some of the so-called trophoneuroses, as entities to symptoms of central origin, that we can scarcely doubt the same mechanism obtains. Observe the changes in the cutaneous and subcutaneous tissues in scleroderma, symmetrical gangrene, trophedema, persistent hereditary edema (Milroy's disease), myxedema, etc. Compare the similarity in the trophic changes of the osseous system seen in tabes, syringomyelia, poliomyelitis, the arthritides, muscular dystrophies, etc.; the manifestations suggest a common mechanism. In the same way vasomotor phenomena, though appearing under quite dissimilar circumstances, present a more or less uniform picture—the analogous vasomotor changes in organic cerebral lesions and those of purely psychic or functional origin. The pallor, cyanosis, redness, depending upon vasodilator and vasoconstrictor influences, like the trophic symptoms, appear in diseases of central localization as well as in peripheral nerve

and sympathetic nerve lesions. This points to a common origin, a fundamental influence. The dependence of vasomotor and trophic symptoms upon the autonomic system, the relationship of the latter to endocrine function, and the influence of both upon the vasomotor and trophic centers within the nervous system, are worthy of critical study.

Clinical observation justifies our recognition of vasomotor and trophic centers in the central nervous system. We may be reasonably certain that these centers are serially represented within the cord and doubtless, too, within the brain; and our observations are leading to the conclusion that there are higher cerebral centers of vasomotor and trophic control. In a case of tumor of the brain observed by us at the Montefiore Hospital, a vasomotor paralysis accompanied a transitory hemiparesis. Another case of brain tumor very recently seen at this hospital showed extensive decubitus in a case of frontal lobe glioma, where the decubitus was not due to local conditions. In many cases of lesion within the central nervous system, the vasomotor symptoms are strikingly transitory. In a case of tuberculoma of the corpora mammillaria, by pressure due to the growth mechanically affecting the function of the pituitary gland, a striking feature was the evanescence of the vasomotor symptoms. There was at times a diffuse redness, with local rise of surface temperature, now of one and again of both sides of the face, and at other times there occurred a peculiar diffuse cyanosis; often a marked cyanosis of one or both lower extremities, quite independent of position, was observed in this same patient.

A cortical center for control, direct or indirect, of the secretory function of the skin is probable and by many writers

accepted. In organic lesions of the brain, anomalies of this function have been reported by numerous observers; for example, in the case of a glioma of the left frontal lobe, unilateral hyperhidrosis of the right half of the face was described. Probably this function has "centers" of control throughout the various levels of the cord. Anomalies of this secretory and excretory function have been, of course, repeatedly described in peripheral nerve injury, and the sympathetic fibers were regarded as the conveying route.

The vasomotor and trophic changes seen in cord diseases, such as tabes, gliosis, etc., in which nutritional modification of the bones, nails, hair and skin are determined by changes within the central nervous system, find analogy in changes seen in peripheral nerve injury. Whether the sympathetic fibers, the motor or the sensory nerves themselves, are the routes affected, the essential control is probably through the ganglion cells of the spinal cord.

The fundamental influence of the nervous system upon the vasomotor and trophic processes is further suggested by a study of peripheral nerve injury with secondary peripheral vascular disturbance. Observations in the world war will doubtless give us valuable data. Stopford made a careful study of vascular and nerve tissue of an amputated leg. The patient was a man of 23, who about a year and a half before the amputation had received a gun shot wound in the thigh. The author concluded from his studies that the endarteritis found in the distal part of the amputated member was secondary to the nerve injury. The irritative effect of the peripheral nerve lesion upon the vascular wall results in changes of the blood vessel, and a narrowing of the lumen follows. Stopford concludes that the vascular changes resulting from the irritative nerve lesion are in part the cause of the trophic manifestations. He makes the practical deduction that prompt relief of this irritation is imperative, in order to avoid

such painful complications as thermalgia, acute decubitus, etc. It is not improbable that Stopford is correct in assuming nerve control of the vessel wall, but it is improbable that he is correct in assuming that the decubitus, thermalgia, etc., are due chiefly to a process of ischemia. There are many other observations, some already related, which bespeak a process within the central nervous system of the nature of what we might term neurometabolic, controlled by tropho-vasomotor centers.

Girou, in an article in *La Presse Médicale*, 14 Nov., 1918, p. 584 (*Causalgies et syndromes douloureux d'origine sympathique*), says that the frequency of wounds of the peripheral nervous system during the war has drawn attention to the physiology of the sympathetic system and to the various clinical features following upon its lesions. These injuries, he believes, have brought out especially the dualism of this system, as a true peripheral nervous system and as a sympathetic system. "To the true peripheral nerves," he continues, "belong voluntary motor innervation and conscious sensibility; to the sympathetic system, the maintenance of the muscular tonus of Est; the vasomotor and trophic innervation of the various tissues, and probably a certain unconscious sensibility. Lesions of the peripheral nerves accordingly manifest themselves by paralyses and by sensory disturbances, anesthesia, hypo- or hyperesthesia, pains; lesions of the sympathetic system, by contractures, trophic and circulatory disturbances. The clinical diagnosis is sometimes much less simple and easy, however, than this formula would seem to indicate. There are sensory disturbances which are referable to circulatory changes due to lesions of the sympathetic system: the vascular medium which surrounds the sensitive terminals of the peripheral nerves having been altered, these sensitive terminals become the starting point of painful sensations. It is imperative to distin-

guish these sensory disturbances from those due to lesions of the peripheral nerves, especially causalgias, for the treatment is altogether different in the two cases; lesions of the peripheral nerves require surgical treatment of these nerves, whereas sympathetic lesions call for surgical measures directed against these lesions."

Neurotrophic fibers (sympathetic) doubtless accompany motor or sensory fibers or both. It is quite conceivable that the fibers of spinal function have their trophic centers throughout cord levels, and are in reality the source of trophism, just as the anterior horn cell is the seat of this function in preserving muscle integrity. The variety of trophic changes in peripheral nerve injury is indeed striking, and invites study and interpretation. In addition to the usual muscular atrophies from disuse or from destruction of the anterior horn cell, there occur bone atrophies, as shown by roentgenologic examination; various skin changes of trophic nature, such, for example, as hyperkeratosis; greater resistance to high degrees of temperature; abnormal growth of nails, or the opposite condition (onycholysis); peculiar furrowing of the nails, anomalies of hairy growth, etc.

In this connection, observations of the vasomotor and trophic changes appearing in the course of such entities as scleroderma, offer a promising field for comparative study. As is well known, this symptom complex is characterized clinically by a change primarily in the normal structure of the cutaneous and subcutaneous tissues. Most varied clinical phenomena of vasomotor and trophic nature may occur during the course of the disease. The affection usually begins as a more or less diffuse swelling of the skin and its underlying tissue, a kind of hard edema, and the process may, as in the case here presented, spread over the entire integument. The affected areas of the cutaneous mantle assume a brownish, wood-like consistence, and finally become immovable upon the parts beneath; the skin grows

leathery. This hide-brown condition may restrict all movements affecting tendons, soft tissues and muscles; the limbs may be fixed at the joints, and the limitation of movement becomes extreme, as in our case. The first or hypertrophic change within the subcutaneous tissue may be followed by atrophy, and it is this atrophic stage which presents the hard, indurated appearance, in some cases resulting in extreme atrophy of the cutaneous tissue with its parchment-like feel. The cartilages of the ears, nose and lids may become hardened; and I have observed, as in the accompanying case, that these changes may be among the first objective findings. In another of our cases at the Montefiore hospital the process was confined to the soft tissues of the lower extremities, both being symmetrically affected. The metabolic changes of the organism may affect the hair and teeth: in the case presented it is to be observed that the hair of the scalp has a wire-like consistence, and we are told that previous to the illness the hair was of a fine and silky texture. The teeth have a peculiar luster, are thin and almost transparent. Our patient appears like a wax figure, the entire body being fixed within its parchment envelope. The scleræ have a peculiar pink hue, and give to the prominent eyeballs a strange mummy-like stare, as they seem to protrude from the waxen setting of the head and face. The sclerodactylism here shown represents an early localization of the trophic pathological process. Together with the cutaneous sclerosis, there is a diminution in the size of the bones of the fingers and osseointerarticular changes of the phalanges, with partial absorption and ulceration. The nature of the changes in the bones as revealed by the roentgenological examination shows symmetry of the process, and is such as to suggest strongly true trophic changes of central origin analogous to those found where endocrine influence is conceded. The process is, as a rule, not painful. Perhaps for this reason and because of the charac-

teristic ulcerative changes of peripheral parts with osseous absorption and changes of the skin and nails, the affection at once suggests central gliosis. As in our case, the affected fingers are often contracted and of a purplish hue, the result of asphyxia not infrequently culminating in gangrene and mutilation. As in the patient presented here, the wrists may become fixed



FIG. 1. SHOWING VASOMOTOR AND TROPHIC CHANGES IN PERIPHERAL PARTS OF EXTREMITIES.

by a kind of ankylosis and the elbows held flexed by the sclerotic process. The muscular atrophy with apparent paresis is due to the contractures of the skin.

Scleroderma has been associated with facial hemiatrophy, with Raynaud's disease, erythromelalgia and other trophic-neuroses. Scleroderma has been frequently observed together with exophthalmic goiter

and with Addison's disease. The anomalies of pigmentation of the skin and mucous membranes so often noted are well illustrated in our patient, in whom the brown pigmentary phenomenon produces a copper colored integument, only here and there showing the normal hue. Apparently visceral localization of the pathological sclerotic process sometimes gives rise to albuminuria, paroxysms of diarrhea, and irregularity of cardiac action. In the latter symptoms we have suggestion of the influence of the autonomic system. Indeed, the likelihood of endocrine origin of the entire process has much to suggest it. Postmortem findings upon victims of scleroderma, who usually succumb to some intercurrent disease or to cachexia, have frequently shown pathological changes in the ductless glands, especially the thyroid and suprarenal glands. The presence of varied symptoms of dysfunction of the endocrine glands is common in scleroderma. A case of exophthalmic goiter was reported by Marinesco in 1913, and is of interest in this connection in that sclerodermic patches developed after treatment of the thyroid by x-ray; pigmentation also appeared. The action of the sympathetic in such cases is clearly suggested. Opothrapy is said to act favorably in scleroderma. In the patient we are presenting, thyroid therapy brought marked improvement for a time early in the course of the disease. That the underlying cause of scleroderma is still obscure is manifest from the various theories propounded as the factors underlying the process. Tropho- and vasomotor-neuroses, sympatheticotonia, endocrine, dysfunction, etc., are all given as the underlying influence. Brissaud and others would seek the lesion in the sympathetic system, while Bruns would place it primarily within the central nervous system, and Cassirer and Curschmann interpret the sclerodermic process as a disease of the vasomotor and trophic tracts and centers. We are strongly inclined to accept the latter theory as the most probable.

The influence of trauma in determining the activity of the process is interesting. Many cases are recorded as having followed a slight injury. One of our patients at the Montefiore Hospital gave the interesting history that the ulcerative process of the hand, followed by sclerodermic symptoms, was directly continuous with a local infection of the extremity. The injury was the result of a prick by a needle. This association of a secondary vasomotor and trophic process with a primary infection is analogous to some of the cases of neuritis with secondary vasomotor changes seen in infected wounds in the war.

The case presented is worthy of study. (Figs. 1 and 2.) The details are as follows: The patient was admitted to the Montefiore Hospital some time after a diagnosis of Raynaud's disease had been made by a physician. She is twenty-eight years old, of Austrian parentage. Her personal history previous to the onset of her present illness gives no facts bearing upon her illness, and there is no hereditary predisposition. The illness causing her present condition began at the age of sixteen. Without prodromes, the initial symptoms, rather abrupt in onset, were swelling and redness, shortly afterwards attended by a subjective feeling of stiffness in the toes of both feet. This was followed by pain in the parts. Progressive stiffness of the smaller joints of the feet, more or less synchronous with a feeling of tenseness of the soft tissue, soon restricted movement in these parts to a degree limiting flexion to very slight movement. All the joints and adjacent soft tissues of the lower extremities rapidly became similarly involved. The lower limbs felt objectively and subjectively cold. The hands became involved some three months later. The fingers gradually stiffened as a result of a process identical with that of the lower extremities. The affection of the fingers suggested an atypical Raynaud's disease. At intervals there occurred paroxysms of digital pallor with pain and dysesthesia in the distal parts.

The blanching in the attacks was followed by congestion and cyanosis. The hands became entirely useless by reason especially of growing weakness and a rapidly progressive contracture of the forearm flexors and a tightening with tension of the skin of the hands. So rapid was this process that within a month it had reached an advanced degree. The symptoms all progressed, reaching such severity as to make the patient practically bedridden.

Two years after the onset the soft tissues about the oral opening and jaws had changed; the tension, due to trophic changes of the skin, restricted the opening of the mouth and moving of the jaws.



FIG. 2. SHOWING CHANGES IN TEETH ESPECIALLY.

Gradually the skin of the entire face and forehead and upper part of the neck grew thin, tense and hard, giving to the touch the smooth, marble-like feel characteristic of this disease.

Teeth.—There is a peculiar luster, especially of the front teeth. (See Fig. 2.) They are of a bluish transparency which gives them an unnatural tinge. This is particularly true of the frontal and lateral incisors. The mucous membranes, especially of the lips, have a dry, tense feel and a pale, anemic color.

Movements.—The general stiffness of the entire cutaneous mantle so affects the movements that in attempting to lift the patient one gets the impression of raising a wax doll.

Hair.—The hair of the head has a dry, wiry feel, quite in contrast to what the patient describes before her illness, when the hair was silky and soft, and the scalp well lubricated with its natural secretion.

Lower Extremities.—The left dorsalis pedis artery pulsates distinctly. At this examination, the extremities, including the hands, were warm and only slightly cyanosed, except the soles. The two feet showed a marked contrast both in appearance and feel; the left was fairly white, showing little cyanosis except about the sole of the foot, where a marked, diffuse duskiness was apparent, emphasized by the contrast brought out by pressure of the soft parts, which, until the blood slowly diffused, left a white, parchment-like appearance. The left foot had a normal warmth while the right was relatively cold and the duskiness about the sole and heel much more marked than on its fellow. That the peripheral vascular supply plays, in some degree, an etiological rôle in producing the local condition is suggested by the fact that the right dorsalis pedis artery conveyed no pulsation to touch. However, the fact that there was a thickening and tenseness of the cutaneous tissue, thus making the recognition of the artery by touch somewhat more difficult, must be taken into consideration in determining whether the pulsation was altogether absent. The posterior tibial artery was not felt in either foot; it is possible that the latter vessels could not be felt

because of the hidebound condition of the skin. This hidebound condition of the cutaneous surfaces was felt well up into the thighs on both legs.

Abdomen.—In the lower abdomen, while the outer layer of the skin had a feeling of tenseness, there was, in contrast to the skin just above it, an appreciable thickness, a distinctly palpable hypertrophy of the cutaneous tissue. The feeling was not of an edematous character, nor was it like subcutaneous fat, but altogether it suggested the hard surface of leather with a somewhat softer composition beneath. The process is probably in part a true hypertrophy of the subcutaneous tissue quite in contrast to the atrophy found in other parts of the skin mantle.

Upper Extremities.—The fore and upper arms showed very marked brownish pigmentation. There was a pronounced growth of hair on the extensor surfaces of the forearms. The brawny induration of the skin together with a peculiar hypertrophic feel to the skin beneath, was likewise well appreciated on the outer surface of the forearms just beneath the elbows; the feel was very much like that of an elephant's hide. The hands, on the contrary, showed a diffuse pink luster, while over the strongly flexed and sclerodactylic upper extremities the most pronounced hidebound condition of the skin was manifest. White, anemic skin surfaces, due in large part to impoverished blood supply and pressure of the underlying osseous surfaces, appeared in both hands, and surrounding these were rings of tissue seemingly about to ulcerate. The radial artery was distinctly felt some two inches above the wrist on both sides, but not at the wrist, doubtless owing to the cutaneous thickening. To touch the vessel seemed full and normal.

Trunk.—Brownish pigmentation was almost diffuse over the trunk, being accentuated in symmetrical patches suggesting segmental distribution.

Labia.—The labia gave a hard, indurated feel.

Hips.—Owing to the ankylosis at the

hip joints, the legs could be separated only about an inch; further local examination, therefore, was impossible.

Anomalies of the secretory function of the skin were interesting. Although the integument about the axillæ showed a marked atrophy, there was at times a striking hyperhidrosis, the odor being of a peculiar character quite unlike the normal hypersecretion.

A minute examination of the central and peripheral nervous system, with especial reference to its sensory involvement, gave entirely negative results. The slight changes present were manifestly due to purely local mechanical conditions.

Acute decubitus is another of the trophoneuroses, perhaps due, as already pointed out by Charcot, to irritation of the posterior central gray matter of the cord. It does not occur in poliomyelitis, even though the lesions be extensive. Déjerine has emphasized the view that nerve degeneration in decubitus is probably the result of the disturbance or the cutting off of trophic influence of the central nervous system, external pressure being merely an occasional exciting cause. Decubitus has been observed, as already stated, in cerebral lesions, and in such cases it occurs as a rule in the middle line or on both sides of the body.

Described as a disease entity in 1898 by Meige, the syndrome tropho-edema is

another condition which suggests the influence of the nervous system in producing trophic changes of the soft tissues. In cases of central lesions, paraplegia and hemiplegia, as well as in distinctly mental affections, tropho-edema has been observed. The influence of the sympathetic system and that of the endocrine glands is suggested by the association of tropho-edema with symptoms indicative of involvement of the pituitary and the thyroid. The local process, with its seat of predilection in the lower extremities, suggests the same pathology as scleroderma and other similar affections of trophic nature.

Perhaps the most valuable type of vasomotor and trophic neurosis for study is scleroderma. The case here reported is a classic form of the generalized type, and is instructive as showing extensive vasomotor and trophic changes. A case such as this studied in the light of the interrelationship between nervous and endocrine activity, with its various changes analogous to those observed in peripheral nerve injury and central lesion, brings us nearer to the view of an evident analogy or even identity of origin of vasomotor and trophic symptoms of seemingly diverse etiology.

There is no question but that true scleroderma as an entity should be classed with the tropho- and vasomotor neuroses.

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POISONING BY HYDROCYANIC ACID GAS, WITH ESPECIAL REFERENCE TO ITS EFFECTS UPON THE BRAIN

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HISTORY.—So. Ri. Age 40, an Italian, had worked for more than ten years as a disinfecter using hydrocyanic acid gas as the active agent. He was admitted to the hospital on July 6, 1918, in the evening. He had entered a house to open the windows and doors after employing the gas in the usual manner. The owner was waiting outside, and after about twenty minutes, having elapsed without his return, others went in and found the patient on the floor breathing heavily and foaming at the mouth. He was brought into the air immediately, and was seen in about ten minutes by the hospital physicians and taken to the hospital. The window of the room was found closed, the door open and the patient's protective mask was off his face. The following facts were recorded in the hospital history:

Family History—Is negative as obtained from his wife.

Personal History.—The patient has been a moderate drinker and smoker. His health was usually good; up to ten years ago he was a coal miner. For ten years he has worked as a fumigator.

Physical Examination on Admission.—The patient is a small, powerfully built Italian, who looks to be about forty years of age. He is breathing stertorously and his skin is quite flushed. His veins are engorged and red instead of blue. There is no evidence of external injury. His breath has an odor which gives one a choking sense,

but is not characteristic of any recognizable poison. He is unconscious and cannot be roused. Head: His scalp is clean and quite bald. Eyes: The pupils are contracted and do not react to light. The conjunctivæ are moderately injected; there is no jaundice. Ears: There is no discharge, and the drums are quite normal in appearance. Mouth: Negative. A chewed cigar stump was found between his cheek and gum. Neck: The neck seems to be drawn down into the chest. The thyroid is not enlarged. No other glandular enlargement, no thrills or murmurs were observed. Thorax: The chest muscles are rigidly contracted and the shoulders drawn up. Both sides of the chest move equally during respiration. The chest is resonant and there is good vesicular breathing throughout. There are many coarse and fine râles heard generally in the chest. Heart: There is no enlargement or displacement. The heart beat is slightly irregular at times. No thrills are felt, no murmurs heard. The heart sounds are strong and the pulse is 120 and of fair force. Abdomen: The abdominal muscles are very firmly contracted. The liver and spleen cannot be felt. No masses are palpable. There is no evidence of fluid. Extremities: The extremities are absolutely rigid. No knee jerks can be obtained and no Babinski reflex, no glandular enlargement, no edema and no deformities are observed.

On admission his temperature was 99 degrees. Pulse 120; respiration 36. During

the first six hours the patient was unconscious, his heart action and pulse became slower, about 70. At midnight he had a tonic convulsion, after which his whole body was in a rigid condition. His pupils were alternately contracted and dilated. He perspired profusely. He emptied his bladder and rectum involuntarily. There were frequent muscular twitchings and his teeth were constantly ground together.

Laboratory Tests. Urine.—Specific gravity, 1022; reaction, acid; albumin, faint trace; sugar, negative; no casts; many urates and uric acid crystals. Blood: Hemoglobin 90 per cent. Red blood cells 6,200,000. Index 0.8, no pathological red cells. White blood cells 15,100, with a differential count of: polynuclear cells 90 per cent; large lymphocytes 5 per cent; small lymphocytes 5 per cent.

July 7: The patient's condition during the night remained unchanged, his temperature began to rise and ranged from 99 to 102.2, his pulse from 68 to 84, his respiration from 20 to 24; his body remained rigid and there were frequent muscular twitchings with profuse sweating with alternating dilatation and contraction of the pupils. The tonic convulsive seizures were periodic, but there was also a constant muscular rigidity.

July 8: The patient had difficulty in swallowing. His body was rigid with frequent muscular twitching and there was profuse sweating. Breathing was irregular and approached the Cheyne-Stokes type. He moved both arms and legs voluntarily, but at long intervals. The muscles were relaxed between the tonic seizures, the knee jerks active, and a doubtful Babinski was observed in the afternoon. The lungs were still filled with fine and coarse moist râles, without evidence of consolidation. The temperature ranged from 101.2 to 104.4, the pulse from 56 to 80, the respiration from 20 to 40. The blood pressure was 128/70. The spinal fluid was drawn under normal pressure, it was very slightly opaque and had 207 cells, of which 45 per cent were polynuclear and 55 per cent

lymphocytes. It contained a very faint trace of sugar and no globulin.

July 9: The patient's condition remained unchanged during most of the day. He presented periods of relaxation and frequent muscular contractions of the whole body. He perspired freely. His knee jerks were absent; there was a doubtful Babinski reflex. His temperature ranged from 102 to 103. His pulse was of good quality at 66 to 72 and his respiration was steady at 24 to 28. He swallowed nourishment for the first time and seemed to recognize that people were about him. His pupils were less dilated.

From the tenth to the nineteenth days of July, the patient's condition did not change materially. His temperature ranged from 101 to 102.8. His pulse gradually rose to 100, his respiration remained in the twenties, his blood pressure was about 125 systolic, 75 diastolic. No consolidation developed in his lungs. His knee reflexes did not return. A Babinski did not develop. His superficial plantar reflex appeared. He perspired a great deal; his urine did not change in character and no acidosis developed. He had a large number of definite attacks every day. Each was characterized by an initial period of pretty complete tonic rigidity and followed by general clonic contractions, associated with a profuse exacerbation of his sweating, and a rapidity in the pulse up to 128. At these times his face was expressionless and he did not respond to stimuli from hearing or sight. Sleeping was fitful and broken both day and night.

During the whole time the patient was observed, he did not speak. He unquestionably recognized members of his family. He would open his mouth when requested, or shake hands. At first these responses were elicited only by demonstrating by gesture what was wanted, but later, after the eighth day of the attack, he would do these acts on being requested. He was not deaf. These voluntary motions were accompanied by a fine tremor and there was a distinct ataxia in moving his arms.

There seemed to be no zones of anesthesia, but there was a lowering of sensibility due to a general loss of mental alertness and a slowness of response to irritation of the skin and of the organs of special sense, which was specially noticed in sight and hearing.

During the last three days of his life the patient grew more stupid and weaker, and finally died of cardiac failure on July 22, sixteen days after his poisoning.

The examination of the spinal fluid was reported on July 18, with the following result: A clear fluid under normal pressure was obtained with 120 cells in each cubic millimeter; of these 40 per cent were polynuclears and 60 per cent lymphocytes. There was a very faint trace of sugar and no globulin. The blood was examined a second time on July 21; the hemoglobin was 90 per cent, the red blood cells numbered 5,500,000, giving an index of 0.8, the white blood cells were 17,700 with a differential count of 75 per cent polynuclears, 23 per cent small lymphocytes, and 2 per cent large lymphocytes; no pathological cells were found. The blood Wassermann reaction was negative.

TREATMENT.—The treatment consisted of meeting the symptomatic indications, cold applications to the head and ice packs for temperatures over 103 degrees, a fluid diet, atropin, alcoholic drinks containing brandy, and at the onset and at the end, hypodermic medication with camphor, digitalis and strychnine.

AUTOPSY.—The autopsy was done by Dr. W. C. Medill and the organs were examined by Dr. C. H. Bailey. The findings were as follows:

External Examination.—Rigor mortis is present; corneas are opaque; the body is cooled to the temperature of the room. It is considerably emaciated. There is no evidence of external violence. Suggillation is present in the dependent parts of the body.

Internal Examination.—The organs of the abdominal cavity are in normal relation to one another. The peritoneum is abnor-

mally dry. The liver is more red than the normal liver. The organs of the thoracic cavity are in normal relation to one another. There is no evidence of inflammation of the pleura. No free fluid is found in the pleural cavities. The heart: The heart is small and weighs 260 grams. It is found in a stage of systole. The heart is filled with bright red blood, no blood clots present. The heart wall is quite firm, and the left ventricle is quite thick-walled but not pathological, since the heart is in systole. The heart valves are all competent. The cardiac muscle has a good healthy appearance but appears to be a little more red than normal. The right auricle and ventricle are markedly dilated. The pericardium shows no evidence of inflammation and contains a normal amount of fluid. Measurements: Tricuspid valve—13.5 cm. in circumference; pulmonary valve—7 cm. in circumference; mitral valve—8.5 cm. in circumference; aortic valve—6 cm. in circumference; left ventricle—1.1 cm. in thickness; right ventricle—0.2 cm. in thickness. The lungs: The lungs are easily removed from the thoracic cavity, no adhesions being present. The right lung is markedly and diffusely anthracotic. At the apex is an old fibrous scar about 1.5 cm. long, containing a few small calcareous nodules. Peribronchial nodes are normal. The lungs everywhere are crepitant, and on section show marked black pigmentation, but no solidification or other abnormalities. The left lung shows three closely set calcareous nodules at the apex. The largest is 2 mm. in diameter; on surface and section, same as right. Larynx and Trachea: No obstruction or evidence of inflammation exists. Ascending aorta is normal, except for a few small patches, up to 2 mm., of ordinary arteriosclerosis behind aortic cusps, especially about exits of the coronary arteries. Spleen: The spleen was rather difficult to remove, because of adhesions. It weighs 95 grams. It is a little more red than usual. The capsule is quite thick. To the upper half of the outer surface a piece of diaphragm is adherent,

by old, firm, fibrous adhesions. The Malpighian bodies and trabeculae are prominent. The pulp is soft, almost fluid. Kidneys: Both kidneys and adrenal glands are present and in normal position. The capsule strips easily; the surface is smooth; scattered over the surface are a few minute, opaque yellow spots, characteristic of fatty degeneration in the convoluted tubules. There are no hemorrhages visible. Cortex measures 6 mm. Markings are plain and organ otherwise appears normal. When the kidney is incised it appears to be deeper red than normal and a little blood drips from it. The pelvis of both ureters is normal in appearance and contains no stones. Bladder: The bladder is empty and shows no evidence of disease. Rectum: This organ contains a number of hard fecal masses. The mucous surface is moderately injected. Prostate: This organ is of normal size, a little more red than normal. No evidence of disease. Stomach and Duodenum: The stomach is found empty. The mucous surface is quite red. The duodenum is also rather red on the mucous surface. The bile ducts are patulous. The gall bladder is normal in size and is filled with bile. There are no stones present. There is no evidence of disease. Liver: This organ seems a little enlarged. Its color is a little brighter than normal. The anterior margin is moderately sharp. The liver is moderately congested, but otherwise normal, except for a dark colored nodule about 3 mm. in diameter on the surface. Pancreas: This organ is in normal position. The duct of Wirsung is patent. This organ is likewise a little more red than normal, but presents no other gross evidence of disease. Esophagus: This organ shows no gross pathologic condition. Intestines: The intestines contain many hard fecal masses. The mucosa of the entire intestinal tract is moderately injected. Voluntary Muscle: Of rather transparent, waxy appearance, otherwise normal. Kidney, spleen and pancreas are definitely of a more brilliant scarlet color than that seen normally from post-mortem oxidation. The liver, heart and lungs do

not show this peculiar coloration, except where smeared with blood. The color where present, however, extends only about 1 mm. below the surface, the underlying tissue being the normal color of the respective organs, the newly exposed surface changing in a few minutes to the brighter hue.

Microscopical Examination. — Lungs showed moderate hyperemia, but no heart failure cells or other evidence of chronic passive congestion. The smaller bronchi showed considerable epithelial desquamation, but practically no exudate. There was also no exudation in the terminal bronchioles or alveoli. A marked anthracotic pigmentation was present throughout; both lungs presented a peribronchial lymph node, marked anthracosis and was otherwise normal. Heart: Muscle and blood vessels were normal. Sudan III stain showed no fatty degeneration. Kidneys showed a moderate congestion, some degeneration and lack of nuclear staining in epithelium of convoluted tubules and were otherwise normal. Sudan III stain shows moderate fatty degeneration of convoluted tubules, and collecting tubules in medulla. Spleen presented a marked congestion of venous sinuses in the pulp, with many mononuclear phagocytes containing blood pigment. Otherwise the spleen was normal. Liver showed a moderate chronic passive congestion with partial destruction of the cells in the centers of the nodules. The liver cells remaining in these situations contained considerable brown granular pigment. There was no increase of connective tissue. The small nodule described in the gross was a typical hemangioma. Sudan III stain showed no variation from the normal amount or appearance of fat. Pancreas was normal. Voluntary muscle was normal.

Anatomical Diagnosis.—Hydrocyanic gas poisoning. Acute dilatation of right heart. Chronic passive congestion, liver, spleen. Acute parenchymatous nephritis. Tuberculosis of lungs. Hemangioma of liver.

Brain.—The brain was examined in the

laboratory of the department of Neurology, and the pathological findings interpreted by Professor Tilney.

The meninges were adherent along the edges of the superior longitudinal sinus. The blood vessels of the pia mater showed more distinctly than is usual because of the bright red color of the contained blood. There was no evidence of meningitis, and the basal cisterns were empty and appeared to be normal. There was no apparent softening of the brain tissue. The surfaces of the brain had a normal appearance; there was no atrophy or flattening of any of the convolutions and the major fissures were not abnormally wide. The basal surface showed no pathological change in the relation of its several divisions and the cranial nerves were all normal in position and relation.

The macrotomic sections of the brain, made in the coronal plane, showed the following: The entire white substance of the brain, in spite of the fact that the usual formalin fixation had been employed, was pliable and much softer than is usually the case. The sections through the frontal lobe showed numerous small hemorrhagic areas scattered bilaterally through the medullary substance. The blood in these hemorrhages was bright red, giving the entire cross section an abnormal pinkish color (see Fig. 1). Sections through the parietal and temporal lobes, while manifesting the same pliability in the tissues, nowhere presented hemorrhagic areas. The basal ganglia, including in this term the thalamus and corpus striatum, were free of any evidence indicative of pathological changes. This is an observation of importance in view of the fact that carbon monoxide poisoning and its allied poison, illuminating gas, have a special toxic predilection for this region of the brain. Sections through the occipital lobe presented a picture similar in all respects to that shown in the frontal region. In this region, also, there were minute hemorrhagic areas of bright red color, confined to the medullary substance and in particular to that portion of white

matter which goes to make up the centrum ovale (Fig. 2).

In the cerebellum, the sections show that this organ had undergone similar hemorrhagic changes. Although the areas were less extensive than in the cerebral hemispheres, the distribution was limited to the medullary substance. The consistency of the cerebellar tissue, which was involved, showed the same friability observed in the end-brain. The appearance of the brain stem was in marked contrast to the gross pathological changes found in the cerebellum and cerebral hemispheres. The mid-brain, pons and medulla showed nothing of a gross pathological nature on section (Fig. 3).

In the investigation of the microscopic lesions of the brain, the following areas were studied: 1. The prefrontal cerebral cortex. 2. The precentral cerebral cortex. 3. The calcarine cortex. 4. The basal ganglia, including the corpus striatum and thalamus. 5. The cerebellum. 6. The mid brain, pons and medulla.

Serial sections were obtained from these regions and stained by the Nissl method—cresyl-violet and eosin-hematoxylin. Pathological lesions were found in the prefrontal, precentral and calcarine areas of the cerebral cortex and in the cerebellum. The superior temporal, the basal ganglia and the brain stem were free from microscopic lesions.

A stratigraphical study of the cortex was made in each area mentioned. The prefrontal cortex showed, under low magnification, the normal seven layers of cells (see Fig. 4), and a normal relation of the neurocytes to the neuroglia; many of the large internal pyramidal layers of cells showed a tendency to an obscuring of outline and an apparent distortion in shape. Upon higher magnifications (see Fig. 5) this feature becomes more distinct and a cloudy swelling may be discerned. The blood vessels in the area showed no alteration and the perivascular lymph spaces, as well as the adventitious spaces of Virchow-Robin, were normal in appearance.

Sections from the precentral cortex of the brain showed marked pathological changes. A low power demonstrated a decided gliosis, not sufficient, however, to interfere with the seven typical layers of this region (see Fig. 6). The most decisive alterations were observed in the inner layer of large pyramidal cells. All of the Betz cells appeared to have been affected in the same manner. They had lost their pyramidal outline. Their cytoplasm was swollen, their nuclei were excentric in position and the Nissl bodies difficult to make out. A distinct neuronophagy was an active process in this region (see Fig. 7). The normal stratification of the cortex in the superior temporal area and the healthy condition of its cells are shown in Figs. 8 and 9. The stratigraphical study of the calcarine area showed the seven layers of cells normal in relation, but with a considerable degree of gliosis, and the solitary cells of Meynert showed a marked condensation of the Nissl bodies and a general swelling. (See Figs. 10, 11.)

In Fig. 12, a characteristic hemorrhagic area in the medullary substance of the cerebral hemisphere is shown. This feature appeared also in the frontal and occipital areas, but was not observed elsewhere. The staining reaction of the blood was abnormal in that the eosin only feebly impregnated the red cells; the white cells seemed unaffected to regular staining. The cerebellum, particularly in the lateral lobes and also to a less extent in the vermis, had the most pronounced pathological change of any part of the brain. This consisted in the almost complete destruction and disappearance of the Purkinje cells. Magnification under low power showed a slight atrophy in the cerebellum. Its primary and secondary folial fissures were less deep than normal and indicated a certain degree of atrophy. The molecular layer was irregular in its depth and to a less extent the same is true of the granular layer. Numerous small hemorrhagic areas were scattered throughout the molecular layer. All of the blood vessels were dilated

and filled with blood (see Figs. 13, 14, 15). In the molecular layer there were many long rod shaped cells of an unusual character, which seemed to be quite independent of blood vessels and were scattered profusely throughout this stratum (Fig. 16). In their disappearance and destruction the Purkinje cells seem to have faded out and to have lost their normal staining reaction (Fig. 17). When recognized at all, they had but a shadowy appearance. This change is notable in the further fact that there was no neuronophagic reaction on the part of the neuroglia, as was seen in the precentral region. This at once raises the question whether the Purkinje cells are more especially susceptible than the other brain cells to the toxic effect of the hydrocyanic acid, or whether they had more readily succumbed secondarily to the blood-vascular changes found in the white substance beneath the cerebellar cortex.

ANALYSIS OF THE SYMPTOMS in the case reported, in their relation to the cerebral lesions.

Initial Coma.—The acute onset was due to the hydrocyanic acid poisoning. The fact that the patient fell when overcome probably saved him from the inhalation of an immediately lethal dose of the gas by landing him in a less concentrated atmosphere near the floor. The explanation of the sudden onset of coma, which lasted twenty-four hours, is to be found in the immediate toxic effects of the poison to inhibit the brain cell activity, not only of the frontal lobes, but at the same time the poison acts indirectly through the blood to paralyze the motor cells of the whole cerebral cortex. When first discovered, the characteristic blood changes, caused by an interference with the normal oxygen exchange of the hemoglobin, had already taken place, as shown by a pinkish color of the superficial veins. This condition did not disappear, although the patient lived sixteen days, and it was a marked feature of the blood even after death.

Phase of General Muscular Rigidity.—

For six hours following admission to the hospital the patient remained unconscious and in an extreme hypertonic condition with arms and legs in extension and the body orthotonous. The pupils were contracted and no knee jerks were obtained. The Babinski reflex was absent on both sides. The hypertonic conditions seemingly must be explained by irritative poisoning of the cerebellum with a continuance of the original disturbance of the frontal lobes, causing unconsciousness. The absence of the knee jerks was probably due to the extreme muscular rigidity which made it impossible to elicit the reflexes. The contracted and immobile pupil was due to cortical irritation.

Phase of Tono-Clonic Convulsions.—

For the remaining fifteen days, the patient had many convulsive seizures of the tonoclonic type in addition to coarse muscular twitchings which affected the arms and legs. Also during this period the extreme hypertonus in the muscles disappeared almost entirely and did not reappear. There was a well marked ataxia with the voluntary movements of the arms which should be referred to the cerebellar lesion. The dominance of the tonoclonic convulsions is interpreted as evidence of cortical irritation reasserting itself over the cerebellar disturbance, and would seem to indicate the rapid destructive changes going on in the cerebellum as shown in the ultimate disappearance of the Purkinje cells. The temporary return of the knee jerks with some exaggeration and a suggestion of a bilateral Babinski, is again evidence of cortical irritation, whereas the subsequent disappearance of these signs would indicate the beginning of a destructive lesion, which, however, did not lead to a complete paralysis. The alternating contraction of the pupils was probably associated with the occurrence of the convulsions, a fact which has already been noted. During the two weeks of his illness the patient had each day a varying number of convulsive seizures. They were always about the same in character and consisted

of a short initial stage of tonic spasm in extension followed by clonic movements of the extremities of short excursion. The pupils were dilated and the sweating was greatly increased during the fit. Each fit lasted something less than five minutes. As described by one observer, the patient had the expression of an advanced case of paralysis agitans. The face was expressionless and the patient would respond readily to any external stimulus. This would disappear between convulsions. One of the cases reported by Cohen¹ which recovered, had three convulsions with tonic spasm, dilated immobile pupils, followed by several clonic convulsive movements, ending with rigid muscles and a period of extreme weakness of the heart with small thready pulse. This frequently recurrent symptom-complex is to be attributed to the direct action of the poison on the cells of the cortical motor area. The fact that he began to move his arms and legs about during the second day of his illness is evidence that there was no immediate destruction of the motor mechanism. It was almost impossible to determine whether these were automatic associate acts or voluntary movements, but the lack of purpose associated them more definitely with the former. On the fourth day he began to regain consciousness, yawning many times, opening his eyes, moving his head from side to side and swallowing. He also seemed to hear, and no lesion is observable in the temporo-sphenoidal lobes of the brain. At this time, some at least of the brain cells had recovered from the primary toxic effect of the hydrocyanic acid, and the subsequent symptoms must be ascribed to the degenerative process in the cortical cells which were affected by the selective action of the poison to create the focalized lesions which have been noted above in the description of the pathological changes. During this stage of his illness, the patient presented the following picture: He was conscious but very apathetic. He could see and hear and there was no demonstrable anesthesia.

He was completely aphasic and remained so until death. There was no true paralysis and no apraxia of general arm and leg movements. He recognized his wife and children and was evidently anxious concerning his own condition. His voluntary movements were accompanied by a distinct but moderate ataxia and there was a fine tremor. The marked cerebellar lesion can explain the ataxia, and the degenerated motor area in the cortex included the centers controlling speech and explains the aphasia.

He improved for nine days in spite of a continuous temperature of about 102 degrees, with a comparatively slow pulse of about 70, and respirations about 20. On the seventeenth day he died, with the symptoms usually attributed to cerebral disease, with temperature rising to 103.5 degrees, pulse 140, respiration 50.

The bibliography of poisoning by prussic acid and its salts is large and dates back in medical literature more than 100 years. It was a more accessible poison and therefore a more popular means of suicide fifty to seventy years ago than it is at present. Most of the literature has to do with the internal use of the drug and there is only a meager list of reports on cases of poisoning from the use of hydrocyanic acid gas. The observers who have investigated cyanide poisoning have all laid stress on the condition of the blood, and none has made any thorough research into the condition of the central nervous system. Geppert¹ has studied this subject thoroughly and formulated the usual cause of death in the rapidly fatal cases thus: "In consequence of poisoning by prussic acid less oxygen is consumed and less carbonic dioxide is formed than is normally the case. The cause for this is that the presence of prussic acid in the body removes from the tissues the power to use oxygen. The poisoning is an internal suffocation of the organs in the presence of an excess of oxygen." Ewald² has brought the theory of Geppert to a modern basis and states that the prussic acid at once destroys the

hemase of the blood and prevents the ferment action of this oxydase blood constituent to further the freeing of oxygen from the hemoglobin. He considers prussic acid as a poison specific for the ferments of the body, especially for hemase and other oxidating blood ferments. The result of this poisoning is the internal strangulation described by Geppert. Kuhlmeier³ gives a careful study of this subject from the point of view of legal medicine, but fails to note any changes in the nervous system as occurring in these cases. His point of view is that of all observers who have been investigated. The case reported here is unusual, both in being caused by the inhalation of the poison and also in having lived seventeen days after the initial trouble. The element of time, therefore, played an important and peculiar rôle and the autopsy findings seem to be unique so far as previous findings have been reported.

The effects of the poisoning by hydrocyanic acid gas can be grouped into three classes or degrees of poisoning dependent upon the dose. In the case of the absorption of a large and overwhelming quantity, the individual is overcome at once and an almost immediate death occurs from the characteristic internal suffocation—the same as results from swallowing lethal doses of poison. In the case of the exhibition of a much smaller dosage, the patient suffers an immediate brain shock with resulting unconsciousness and extreme cardiac weakness, and after a varying time he recovers completely without permanent injury. Such cases may or may not present the characteristic fits which are typical of hydrocyanic poisoning. Cohen,⁴ in 1916, reports two cases both with recovery, one with fits and one without this symptom, and Regnauld⁵ reported in 1852 a similar case of attempted suicide from hydrocyanic acid gas which had tonic muscular spasms during 4½ hours, and completely recovered in forty-eight hours.

The case reported in this article is intermediate between the two extremes. The

patient survived the initial brain-shock and the selective quality of the poison caused focal lesions in the brain, which seem ample to explain the characteristic

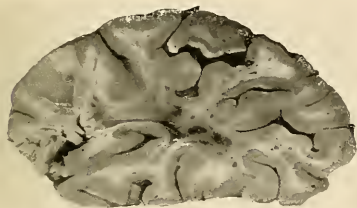


FIG. 1. HEMORRHAGE IN FRONTAL LOBE.

symptom complex which has been described. The symptoms are an initial unconsciousness followed by a period of recurring cerebellar seizures; of aphasia of motor type; of a febrile action with pulse and respiration lower than usual, with the degree of temperature reaction observed, such as is dependent on a cerebral lesion; and of a terminal death with cardiac weakness and rising temperature.

Corlette² reports, in describing the use of hydrocyanic acid for fumigation against insects, that this method has been used only in the United States, Australia and South Africa. In the United States it has been used against the parasitic diseases of

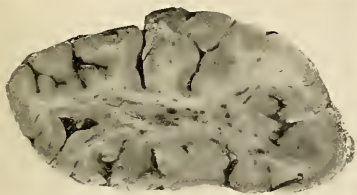


FIG. 2. HEMORRHAGE IN OCCIPITAL LOBE.

citrus fruits, and has therefore been largely employed in Florida and California. Four deaths have been collected by Corlette and McIntosh as having occurred

in Australia, and he states that no deaths have been reported in California. No deaths have been recorded either in New York City, or to the New York State Board of Health, prior to the case here reported, in spite of the fact that the method of fumigation has been used for at least ten years, although in a limited application.

McIntosh⁶ reported a case of poisoning by hydrocyanic acid gas which lived four days and a half. He was poisoned by about three minutes' exposure in a room being fumigated by the gas. When discovered he had stopped breathing, was pulseless and quite unconscious. A pinkish frothy mucus constantly came from the mouth. He gradually recovered with artificial

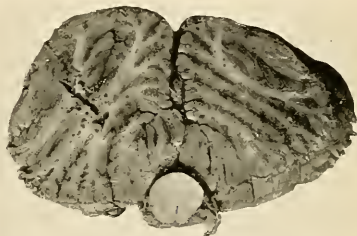


FIG. 3. HEMORRHAGIC AREAS IN CEREBELLUM.

respiration, with a slowly uprising pulse, respiration and a low temperature, labored breathing and a sluggish pupil reaction. After some hours he had his first general convulsive seizure described as resembling that of tetanus, lasting five minutes, and recurring at intervals of ten minutes. The spasms gradually became less frequent and severe, but though diminished in severity, continued until death, four and a half days after the accident. A lumbar puncture drew 4 c.c. of fluid, which showed no departure from the normal. The knee jerks were sluggish, the Babinski reflex positive on both sides, and there was no ankle clonus. He died with a rising temperature, of cardiac weakness. The post-mortem examination showed a slight con-

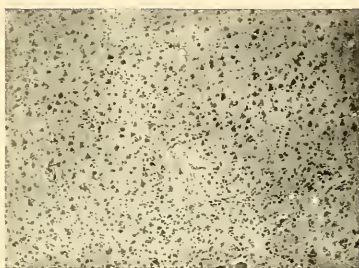


FIG. 4. PREFRONTAL CORTEX.

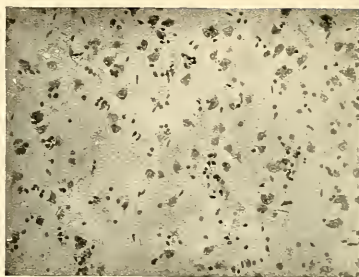


FIG. 5. PREFRONTAL CORTEX.

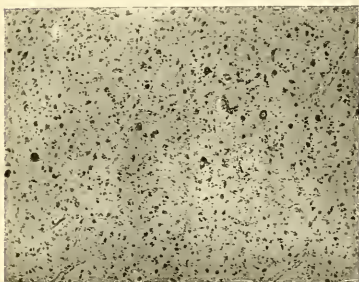


FIG. 6. PRECENTRAL CORTEX.

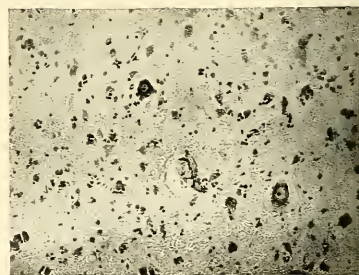


FIG. 7. PRECENTRAL CORTEX.

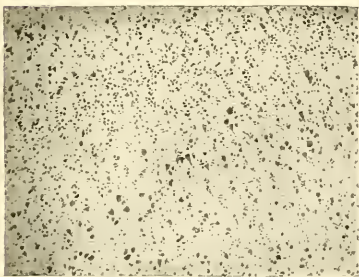


FIG. 8. TEMPORAL CORTEX.

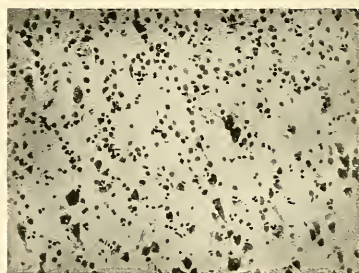


FIG. 9. TEMPORAL CORTEX.



FIG. 10. CALCARINE CORTEX.

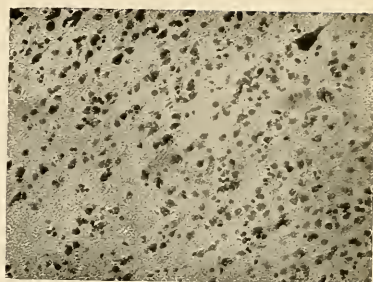


FIG. 11. CALCARINE CORTEX.

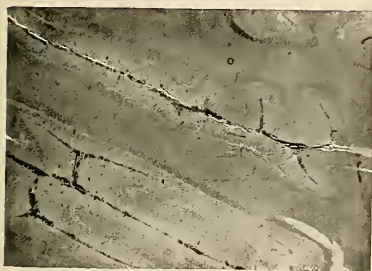


FIG. 13. CEREBELLAR CORTEX.



FIG. 14. HEMORRHAGE IN CEREBELLUM.

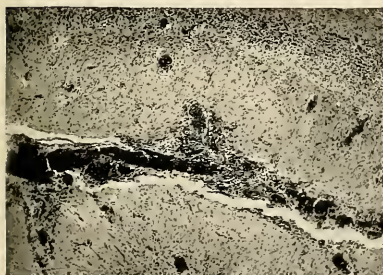


FIG. 15. HEMORRHAGE IN CEREBELLUM.

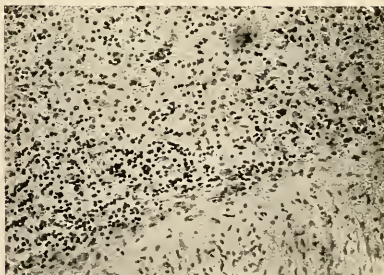


FIG. 17. DISAPPEARANCE OF PURKINJE CELLS.

gestion of cerebral convulsions and chronic pyelitis. The whole brain was examined, there was no abnormality apparent to the naked eye. A microscopic examination of the brain was not carried out.

Mittenzweig⁷ reported in 1888 two cases of poisoning from inhalation of gas of hydrocyanic acid, which present an entirely different picture, and one which can be explained only by presupposing a per-

ness of tendon reflexes, lessening of electrical reaction in skin, muscle and nerves, sleeplessness, loss of appetite and anemia. Acute symptoms at the time of the poisoning consisted of irritation of the respiratory tract and of the intestine; headache, dizziness and a feeling of faintness; weakness of the heart and disturbance of motion and sensation. Both cases resulted from poisoning by slight doses of the gas. One, a house-



FIG. 12. HEMORRHAGE IN CENTRUM OVALE.



FIG. 16. HEMORRHAGE IN CEREBELLUM.

manent, but not fatal injury to the central nervous system. One case is given on the authority of Dr. Martin of Munich in "Friedrichs Blätter für gerichtlicher Medizin und Sanitätspolizei." XXXIX. Jahrgang, I. Heft, and the second from his own experience. Both cases resulted in a chronic and permanently incurable condition of ill-health characterized by pains in head and back, extreme exhaustion, shuffling gait, weakness, feeling of cardiac depression, lowered sensibility in skin, weakness in the voluntary muscles, with moderate reaction of degeneration, disinclination to stand or walk for any length of time, weak-

ness of tendon reflexes, lessening of electrical reaction in skin, muscle and nerves, sleeplessness, loss of appetite and anemia. Both cases remained in the described condition of ill-health permanently. The diagnosis was left by Mittenzweig as an "Intoxication Neurasthenia," because he was unable to explain the condition on a physiological basis. Similar neurasthenias have been described after acute poisoning by carbon monoxide and nitrobenzin;

and the alcoholic symptom syndrome of Korsakov must also be borne in mind.

The case described in this article seems to warrant the conclusion that hydrocyanic acid gas must be added to the list of poisons which may cause a selective lesion in the nerve cells of the higher brain centers. The well-understood lesion in the blood caused by hydrocyanic acid is usually so rapidly fatal that this equally characteristic lesion in the central nervous system cannot develop, because a very considerable period of time is necessary for changes of this nature to manifest themselves in sufficient degree to attract attention either from an examination of the gross specimen of the brain or even from the ordinary microscopic examination of such tissues. The unusual length of time that this patient lived after his initial poisoning would seem to have permitted the description of a new fact in the pathology of gaseous poisons in general and of poisoning by hydrocyanic acid gas in particular.

In conclusion it is a pleasure to record the earnest helpfulness and enthusiasm of Drs. Medill, Bailey and Tilney which have made the report of this case possible.

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EPIDEMIC ENCEPHALITIS.

A PRELIMINARY CONSIDERATION OF SOME OF ITS PROMINENT CLINICAL AND PATHOLOGICAL MANIFESTATIONS

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The epidemic appearance of an unusual disease which affects the nervous system profoundly has, during the past year, been the subject of much interest and inquiry both in England and in this country. From the fact that its most conspicuous clinical feature is prolonged somnolence it has acquired, in the lay prints, the perhaps not unjustified, if somewhat confusing, designation of "Sleeping Sickness."

Economo² devised the term "Encephalitis Lethargica" in describing a similar epidemic in Vienna during 1917, but as Kinnier Wilson¹⁰ has pointed out, this nomenclature is inaccurate, since it is the patient who is lethargic and not the encephalitis; so that to label it "Epidemic Encephalitis" is less open to criticism if not as picturesque.

The chief characteristics of the disease are that:

1. It tends to appear in epidemic form.
2. It produces prolonged somnolence or a state closely resembling lethargy.
3. It gives rise to palsies of the cranial nerves.
4. It causes a marked asthenia.
5. It is pathologically based upon an acute encephalitis.

Its pathogenesis is a little understood and much debated question.

From the very nature of the lesion, an inflammation of the brain, it might be predicted that there would be a multiplicity in the cerebral symptoms. The remarkable fact is that the clinical picture presents such distinct generic definition in spite of the specific variations to which

it is subject. It is especially to these variations, or subgroups of the disease, that attention is here directed, not only as a matter of classification but also as an opportunity to discuss the significance of the disease in some of its relations to certain aspects of cerebral physiology.

Of twenty cases of epidemic encephalitis which have come under the observation of the authors, eight fairly well-defined subgroups or types have been recognized, as follows:

1. The lethargic type.
2. The cataleptic type.
3. The paralysis agitans type.
4. The polioencephalitic type.
5. The anterior poliomyelitic type.
6. The posterior poliomyelitic type.
7. The epilepto-maniacal type.
8. The acute psychotic type.

Each subgroup is described because of some distinctive feature in its development and course. It would almost seem advisable to include in this list another type, the recognition of which depends rather upon the extremely early age of incidence than upon any particular clinical feature. At all events, the occurrence of the disease in infants during the first month of life is perhaps a striking enough character to justify a tentative group in the new-born, encephalitis neonatorum.

I. LETHARGIC TYPE.

In this type of epidemic encephalitis the predominant clinical feature is the lethargy or profound somnolence which the patient

manifests. This condition can hardly be described as a coma or stupor, since it is possible to arouse the patient momentarily out of the deep sleep into which he has fallen, to gain his intelligent coöperation for a brief period in which there is evidence of little or no mental clouding, after which interval he lapses into deep sleep again. In not a few such cases the lethargy continues for a number of days, even until the patient dies, without apparent further involvement. In most instances, however, the lethargy is complicated by the appearance of cranial nerve palsies, most commonly paralysis of the oculomotor group, while in those cases which make a more or less complete recovery, an extreme asthenia develops early and is a persistent symptom.

The first case illustrative of the lethargic type is selected as an especially good instance of this variety of the disease, and also because it has been possible to study the entire brain stem in complete serial sections.

CASE I. Mrs. de la C. A married woman, aged 36, whose previous personal and family history had little bearing upon her final illness. Four weeks prior to the onset of the disease she suffered from what had been regarded as an influenza bronchitis. She was not seriously ill at any time during this affection and made what appeared to be a complete and satisfactory recovery. One morning after an apparently good night's rest, she failed to rise at her usual hour, and in fact, showed no inclination to do so. This at once attracted the attention of her family, since she had always been an energetic and efficient housekeeper. It was found, however, that in spite of all efforts to arouse her, she would fall asleep again after opening her eyes for a few seconds and responding intelligently to questions asked her, without seeming to realize anything except that she was very tired and wanted to sleep. In the brief periods in which she was awake and conversed with the family her replies to questions indicated

that she was suffering no pain and was not conscious of any distress. When seen by her physician she was still in the same deep sleep, breathing regularly and with normal heart action. The temperature taken by mouth showed no elevation. This somnolence continued for two days interrupted only by brief periods during which she was sufficiently aroused to take small quantities of food and fluid, both of which were swallowed in a normal manner. Her almost trance-like condition aroused the suspicion in her physician's mind that the patient was suffering from hysteria, and upon this basis he sought some explanation in the more recent occurrences of her life. It developed that there had been some slight misunderstanding between the patient and her husband, but upon careful investigation this proved to be nothing more than many other similar and customary differences which had occurred in the past. After two more days of increasing somnolence, the theory of hysteria was finally abandoned and the patient was seen by a neurologist. At this time it was still possible to arouse the patient for an instant or so, but immediately she would lapse back into a profound and quiet sleep. When asked to open her eyes, she attempted to comply, but was unable to elevate the lids, in spite of the fact that she clearly understood the request to do so. Swallowing had become difficult and unless fluids were carefully given, would cause paroxysms of coughing and some regurgitation of the fluid through the nose. When requested to protrude the tongue, the patient, although appearing to understand what was said, was unable to move the tongue in any direction. On several occasions she attempted to speak but her voice sounds were feeble, husky and indistinct. When her lids were elevated by the examiner, there appeared to be a slight double external strabismus and a widely dilated pupil which did not react to light upon either side. The pulse and respiration were normal and the temperature by rectum was 101 degrees.

The remainder of her neurological examination showed that all of the deep reflexes were present, including the jaw, pectoral, biceps, triceps, radius, ulnar, wrist, patellar and Achilles, and equal on the two sides. Of the superficial reflexes, the upper and lower lateral abdominals were present and equal, right and left. There was no Babinski, Chaddock, Oppenheim, Gordon or Schaefer.

No abnormal involuntary movements, such as tremors, twitchings or spasms, were observed in any part of the body, nor were they observed or reported at any time during the disease.

No estimate could be made of the muscle strength, although the patient was able to and did move all four extremities and occasionally turned from one side to the other. The electrical reactions were not taken. Myotatic irritability was normal.

It was impossible to obtain any satisfactory estimate of general somesthetic sensibility, because of lack of coöperation on the part of the patient. It did appear, however, that she had perception, especially to the pin-point and tubes containing hot water. She showed evident displeasure when stimulated in this way or by over-extension of the fingers and toes.

Concerning the cranial nerves, no conclusion could be drawn with reference to the special senses although she seemed to see when the eyelids were opened and understand what was said to her. The eye-grounds were normal; the pupils were widely dilated but did not react to light, either directly or consensually. The drooping of both lids was evidently due to a definite ptosis, and the existence of a slight degree of double external strabismus has already been noted. There was no nystagmus or other involuntary eye movements. In the later stages of the disease the jaw muscles seemed to have become paralyzed, as the lower jaw dropped and the mouth gaped widely. There was distinct weakness of both sides of the face, while the difficulty in deglutition and

phonation, together with a paralysis of the tongue, have already been mentioned.

At the end of the fourth day of her illness she was transferred to the hospital for further investigation. Her temperature had risen to 103° by rectum, her respiration was shallow, and her heart action weak. The systolic blood pressure registered 100 mm. Hg.

Examinations of the blood and spinal fluid were entirely negative; the spinal fluid showed no increase in cells and no globulin, and was negative to the Wassermann test.

Deglutition became increasingly difficult so that it was necessary to feed and stimulate by rectum. During her second day in the hospital, respiration grew progressively more shallow and seemed to be interfered with by some obstruction in the upper respiratory passages. It appeared advisable to make a laryngoscopic examination in order if possible to determine the point of obstruction. This examination revealed a bilateral total paralysis of the vocal cords. Several hours after the laryngoscopic examination, respiration ceased and finally after a few gasping attempts to breathe, the patient died of respiratory paralysis.

POST-MORTEM REPORT ON THE BRAIN

The Meninges.—The dura mater covering the convexity of the brain and also upon the base was normal in its appearance. The pia mater, however, especially that covering the basal surface, showed a considerable degree of injection, while upon the convexity nothing considered pathological was noted.

Surfaces.—All surfaces of the brain were normal in appearance, there being no evidence of hemorrhage, softening or atrophy.

Macroscopic Sections.—Sections of the brain in the gross showed nothing pathological in the cerebral or cerebellar hemispheres. The entire brain stem was intensely injected and had a pinkish appearance suggestive of recent inflammatory

reaction. This was particularly true of the medulla oblongata, the midbrain and the thalamus.

Microtomic Sections.—Upon microscopic examination the pia mater covering the medulla oblongata showed considerable injection and there was a moderate degree of round cell infiltration, not, however, comparable to the condition seen in lethal cases of acute anterior poliomyelitis. Two of the cranial nerve nuclei, the nucleus hypoglossi and nucleus ambiguus, were specially investigated; both of these areas showed distinct cellular changes and nearly all of the cells in both nuclei appeared to be pathological. These alterations varied from a moderate degree of cloudy swelling with eccentric nucleus and obscurations of the Nissl's bodies to a fairly advanced chromatolysis in which the more central layers of tigroid bodies had disappeared. A slight degree of neuronophagia was manifest, especially in the hypoglossal nucleus. Ameboid glia cells could be observed collected around many of the motor cells of the nucleus which were in a more advanced stage of degenerative change.

The blood vessels in the medulla oblongata presented an interesting picture; both veins and arteries were much engorged, but the hyperemia was more pronounced upon the venous than on the arterial side. Numerous small hemorrhagic areas were observed, especially around the smaller veins, and a fairly well defined cellular infiltration was found in the adventitial lymph spaces which contained accumulations of leucocytes and plasma cells. A slight degree of gliosis was discernible in and about the nucleus of the hypoglossal and vagus nerves.

Pons Varolii.—This portion of the brain stem also showed a slight degree of infiltration in the pia mater. The nucleus abducentis was especially studied, and here, as in the case of the medulla, the large motor cells showed evidence of acute change, varying from intense cloudy swelling to a moderate degree of chromatolysis. In a few instances, the cell bodies showed

vacuolization. The vascular picture was similar to that observed in the medulla and was characterized by numerous small hemorrhages apparently in relation to the veins, all of which were intensely injected. The tegmental portion of the pons presented a slight degree of gliosis, but this could not be made out with certainty in the basal portion.

Midbrain.—More pronounced, perhaps, in this region than in any other portion of the brain stem were the changes already described. Not only did the motor cells in the third nerve nucleus and the cells in the red nucleus show such changes as cloudy swelling and chromatolysis with a moderate degree of neuronophagia and vacuolization, but the hemorrhagic areas were more numerous, and in many places the vessels showed thrombosis with a tendency to necrotic change in the adjacent tissues. This was particularly true of the region of the midbrain immediately about the substantia nigra. Similar hemorrhagic and thrombotic changes were observed in the thalamus, hypothalamus and corpus striatum.

A summary of the pathological alterations observed in this case justified the diagnosis of acute polioencephalitis. The nuclear changes in the medulla explained the paralysis of the tongue and the final failure of cardiac and respiratory action. In the same manner the alterations in the midbrain about the nucleus oculomotorius furnished the cause for the oculomotor palsy, while there is some ground for the belief that the changes in the thalamus and about the aqueduct of Sylvius may be held responsible for the prolonged and deep somnolence.

The pathological changes in this case are shown in Figs. 1-6.

CASE II. The second case of the lethargic type of the disease is that of a physician forty-three years of age who suffered from influenza in November of 1918. After recovery from this infection, he was busily engaged in practice until March 2, 1919. Much of his time was given to caring for

patients suffering from influenza during the severe epidemic of this disease which prevailed in this city during the winter. On March 2, 1919, he developed a temperature of 104° which continued for one day, and varied for the next three weeks between 100° and 102° . During the first part of this period he was very restless and unable to sleep and then he began to yawn and become very drowsy, sleeping most of the time; he would even fall asleep when conversing with his friends or physician. His expression became glassy and fixed. He could be aroused to take nourishment for a few moments and although he seemed oriented and in complete contact with his environment, his entire attitude at these times was one of indifference and apathy. He remained in bed for three weeks until the fever disappeared and following this was confined to the house for another week. Mentally he was very much depressed, but in addition to a marked degree of salivation, showed little in consequence of his recent illness. Upon advice, he took a trip to the mountains which seemed to do him little good; the depression and salivation still remained and the tendency to yawn returned again. He noticed an oily secretion from his forehead, and his skin would show a red tache on even the slightest stimulation. There was an unnatural flush over each malar prominence and his face had a glassy and fixed expression. Upon returning to practice he noticed that he was troubled with much nervous unrest, a feeling of disquiet in his legs. This made it necessary for him to move his feet about constantly whenever he sat down. This sense of disquiet was further augmented by the development of a peculiar contraction of the muscles of the big toe on the right foot, which occurred spasmodically many times a day whether he was walking or at rest. Latterly he has complained of a slight tendency towards festination, so that while the case may be considered as representative of the lethargic type, it seems to tend in its late consequences towards some of the features of the Parkinsonian syn-

drome, especially the wax-mask, the disquietude in the legs, and the tendency towards propulsion.

CASE III. P. E., Presbyterian Hospital, No. 40,374. Male, fifty-two years of age, driver by occupation. On January 1, 1919, the patient developed a fever, became markedly irrational and complained of severe, constant headache. His history showed no previous illness, either recent or remote. He had not suffered from influenza. After a period of marked restlessness and insomnia lasting three days after admission into the hospital, he gradually became more and more somnolent and remained in a stage of lethargy for three weeks. During this time he could be aroused and for a moment or two would respond to questions and follow simple instructions. He took his food well and would even sit up in bed if instructed to do so. Immediately after being aroused, however, he would lapse back into a somnolent condition, in which he would remain almost without a movement of his limbs for a number of hours. Both arms and legs were held in a semiflexed position most of the time but it was possible to extend them without much force, the muscles seeming to be in a slight degree of spasticity. All the deep reflexes were active and equal on the two sides. There was no clonus and no Babinski or any other pathological reflex observed. On January 14, 1919, the right pupil was larger than the left and was fixed to light stimulation. There was also a right divergent strabismus but the fundoscopic examination showed nothing abnormal in either eye.

Upon testing the vision, it was found that there was a diplopia on looking both to the right and to the left. This was considered indicative of a partial paralysis of the third nerve on the right side.

The following laboratory records are significant:

January 2, 1919. Spinal fluid, 5 c.c. withdrawn, slightly tinged with blood. There was no increase of pressure. The fluid contained 23 lymphocytes and 5

polymorphonuclear cells. No organism was detected in the smear; the culture of the fluid was negative, as was also the Wassermann reaction.

Blood culture on the same date was sterile; the stools showed no blood, bacillus typhosis or bacillus dysenteriae. The blood Wassermann test was negative and the blood count showed white blood cells 12 700; polymorphonuclear cells 83 per cent.

January 10, 1919. Blood count normal. X-ray of the skull negative.

January 11, 1919. Spinal fluid, 15 c.c. of clear fluid obtained under no increase of pressure. Cell count shows 12 lymphocytes and 1 polymorphonuclear cell. Globulin was negative, culture was sterile, Wassermann test negative.

A guinea pig injected with the fluid obtained from the lumbar puncture remained normal.

Subsequent Wassermann tests upon the blood and spinal fluid, as well as blood counts and chemical investigation of the blood, revealed nothing pathological.

The patient remained in this stuporous condition until the 1st of February, 1919, but at this time was fairly rational when questioned, although he occasionally attempted to get out of bed.

On February 3d the patient became more acute mentally and was quite rational. His somnolence had almost entirely disappeared; he sat up in bed and ate with apparent enjoyment. During the following week he gave the impression of a man suffering from some mental impairment without fever or other indication of acute illness. He had so far recovered by February 14, 1919, that it seemed advisable to discharge him from the hospital. Ultimately he made a complete recovery.

CASE IV. J. B., age fifty-nine years; a butcher. In the fall of 1918, the patient suffered from a rather severe attack of influenza from which he made a fair recovery. In the early part of December, however, he suddenly became ill again while carrying on his day's work. He com-

plained of pain in the head and great fatigue which made it impossible for him to remain at his business. He came home and went to bed. In a short time he fell into a deep sleep and with the exception of an occasional low muttering delirium remained in this condition for the following four weeks. His temperature at the onset was 102.5°, at which level it remained with very few and slight exacerbations during the remainder of his illness. At the end of two weeks, his continued lethargy alarmed his family and several consultations were requested.

At this time, neurological examination showed that all of the reflexes were normal although slightly more active in the lower extremities than in the arms. They were equal upon the two sides and no pathological reflexes were observed. The tone of the muscles throughout the body was slightly increased but there was no stiffness of the neck. The patient remained immobile in one position for hours at a time; his breathing was normal and regular; his pulse and blood pressure good. The sensory examination was rendered impossible because of lack of sustained coöperation on the part of the patient. He could be aroused from his lethargy sufficiently to answer a question intelligently and then would lapse into sleep again or perhaps into a low muttering delirium.

The cranial nerves showed a slight degree of ptosis in both eyes; both pupils were widely dilated and reacted but sluggishly to light, with a slight external strabismus of the left eye. The blood and spinal fluid were negative to all laboratory tests.

At the end of four weeks, he began to emerge from his lethargy and manifest interest in his surroundings. He has not, however, within the past four months, recovered sufficiently to return to his business. In fact, his mind seems to be blank and he sits, most of the time, in an apathetic and indifferent attitude, considerably disoriented and without initiative. His oculomotor palsies have entirely disappeared.

This case is illustrative of the lethargic type of the disease in which the cerebral cortex appears to have suffered severe injury as evidenced by the slow psychic improvement in the patient.

The characteristic clinical features in this lethargic group of cases is the pronounced lethargy which was common to them all, together with an elevation of temperature. Three of the four patients had previously suffered from influenza within a relatively short time of the onset of the encephalitis. In three of the cases, cranial nerve involvement causing oculomotor and bulbar palsies occurred. Only one of the cases terminated fatally and that after a rapid course of six days' duration. Of the patients who recovered, one showed no ill effects of the disease, one had a prolonged asthenia, and the third is giving some evidence of a tendency in the direction of a Parkinsonian syndrome.

II. CATALEPTIC TYPE

The two cases of this group present a striking similarity. Both were young girls, one eleven years of age, the other sixteen years old.

CASE V. R. Z., female, sixteen years, operative. The patient was previously a healthy, robust girl who, upon returning from work December 9, 1918, complained of a severe pain in the index finger of the right hand which came on during the afternoon. Later in the evening, the pain spread to her right arm, where it persisted for several hours only to disappear as suddenly as it had come, and leaving behind it a slight paralysis. At this time she suddenly became irrational in her actions. For a short time she was quite violent, even requiring restraint and sedative treatment. Her family believed she had "gone insane." Subsequently she became quieter and fell into a deep sleep. Her temperature showed a slight elevation of 102° with a corresponding quickening of pulse and respiration. She continued to sleep the following day and never from that time throughout the

remainder of her illness, which lasted eight weeks, did she again open her eyes. For some days she lay in this lethargic condition, able to move her body from side to side and having some voluntary control of her muscles. At the end of the first week of her illness, she manifested a general rigidity in her entire body which, however, was peculiar in the fact that the limbs could be easily moved from one position into another. There was no stiffness of the neck or other evidence of meningeal involvement.

Although seeming to be in deep sleep, the patient gave evidence of hearing and understanding all that was said to her when aroused. She was unable to make any adequate response in reply to questions or directions. If asked to protrude her tongue she would make a feeble effort to do so. When told to flex the forearm upon the arm, she made evident attempts in this direction but seemed unable to carry out any voluntary act. Both eyelids remained closed and all her own efforts to elevate them were unavailing.

Neurological examination at this time showed the patient lying supine in bed, eyes and mouth closed, forearms slightly flexed on arms, the latter being adducted, fingers partially flexed, legs flexed on thighs, the thighs slightly flexed upon the pelvis. This attitude gave her the appearance of "an effigy on tomb" as Hall has already described this appearance.

She made no movements; her deep reflexes were present and active on the two sides but there was no apparent pathological change in them.

The superficial reflexes were present and active on the two sides. There was no Babinski or any modification of this reflex. The muscle tone was markedly increased, showing a pronounced, inflexible rigidity. The muscle tissue itself was firm and tense but showed no disposition toward cramping.

The patient was unable to coöperate, hence all sensory tests were omitted. There was no response to tests for the sense

of smell, sight or hearing and for this reason it is impossible to form judgment concerning the special sensory status. The drooping of both upper lids already referred to was due in part, at least, to an oculomotor ptosis but the impression was also conveyed that there was a certain increased rigidity in the palpebrarum orbicularis muscles which tended to hold the eyelids closed. The eyegrounds were normal. Both pupils were widely dilated and failed to react to light. There was slight external strabismus of the left eye.

The masticatory muscles held the jaws partially closed. There was no facial palsy, but the facial muscles seemed stiff and fixed, giving the patient an unchanging expression. The winking reflex was absent.

The glosso-pharyngeal, vagus and spinal accessory nerves gave no evidence of pathological involvement, although at times the patient had some difficulty in swallowing.

In general appearance the tongue was normal and there seemed to be no hypoglossal involvement. In so far as might be judged from the limited coöperation on the part of the patient, the mental status was normal. When aroused, she heard and understood what she heard, and on several occasions showed that she was keenly alive to the purport of conversation carried on about her. This was particularly evidenced when the remark was inadvertently made in her hearing that she was very ill and would probably not recover. Immediately tears began to stream down her cheeks, a reaction which could not be regarded as purely coincidental.

Her family history was negative except for epilepsy in an older sister and a partial hemiplegia in her father which has already cleared up.

The neurological examination showed that there was no true paralysis, since the patient could make faint indications of movements in all parts, her efforts being very slow and of limited excursion. She was unable to raise her head from the pillow and seemed to have no power in the

back muscles. When any movement was attempted, a coarse tremor developed, which had a moderate intensity and began in the extremities whence it spread to the rest of the musculature. Repeated examinations of the reflexes failed to disclose anything that was considered pathological in this regard and the apparent paralysis was considered as due to the increased muscle tone rather than to any direct pyramidal tract involvement.

Of the cranial nerves, the oculomotor group, including the third, fourth and sixth nerves, showed a right ptosis and the right pupil somewhat smaller than the left.

The trigeminus was apparently normal in both its sensory and motor divisions.

Laboratory Tests.—The blood and spinal fluid were investigated several times. In each instance the blood proved negative, both in its count and general morphology. The Wassermann was negative, as was also the blood culture. The spinal fluid was clear and showed no increase in pressure. It contained three lymphocytes, no globulin and the Wassermann reaction was negative. At no time did the urine show any pathological changes.

The patient's mental condition remained unchanged. Gradually her stupor became more profound until it was impossible to arouse her. This change was attended by a gradual tendency to increased temperature accompanied by an elevation of the pulse which fluctuated between 120 and 130 for some days before death. The temperature is shown in chart I. After an illness of forty-seven days the patient passed into a terminal coma and died. No autopsy was obtained.

CASE VI. J. B., age eleven, school girl. This girl had always been a healthy and bright child until December, 1918, when she suffered from a severe attack of influenza. At the same time her mother and two other sisters had this disease. The mother and one of the sisters died of it, but the patient made a complete and rapid recovery. She returned to boarding school in

the early part of January. On January 30, 1919, she was seized with what was considered a nervous chill, followed by a rise of temperature, sleeplessness and a coarse, choreiform type of twitching which manifested itself in all the muscles of the body. Three days later she became stuporous and passed into what was considered to be a coma. Upon admittance to the hospital, her position in bed was described as that of "an image," for she did not move her limbs; her eyes remained closed and it seemed impossible for her to express her wishes by any sign whatsoever. Although in this seeming deep lethargy it was apparent to those about her that she understood most of what was transpiring, for when instructed to squeeze the doctor's hand when she meant to answer "yes," she would on several occasions make feeble pressure in this way.

The neurological examination showed a patient, lying in bed upon her back, eyes closed and cheeks flushed, breathing quietly, with arms, legs and the rest of her body held rigidly in a fixed position. The muscular status showed a marked hypertonus which made passive flexion and extension quite difficult. If placed in a given position, the arm or leg would remain as placed without any attempt on the part of the patient to change it. There was a slight carpopositis with moderate flexion of all fingers of both hands, as well as a pedoptosis with a tendency for the toes to be held in plantar flexion.

The reflexes, however, were all normally active and equal on the two sides. The threshold of both patellar reflexes was twelve ounces, which may be regarded as up to normal standard. There was no clonus at the ankle or patella, neither was there a Babinski nor any confirmatory sign of the same.

The conclusion drawn from the reflex status of the patient was that, although she was markedly hypertonic in her muscles, this was not due to a pyramidal tract lesion, but should be regarded as a defect in the extrapyramidal system.

When passively elevated, both arms would show a coarse oscillation or tremor and the same was true of the lower extremities. Upon passively opening the mouth, a coarse tremor was noted in the tongue. There were no abnormal, involuntary acts reported or observed and no abnormal associated movements could be elicited.

Tests for somesthetic sensibility were unavailing, since the patient could not cooperate in the examination.

The cranial nerves accessible to approach, showed a bilateral slight degree of ptosis and some rigidity in the orbicularis palpebrarum muscle. The third, fourth and sixth nerves could not be properly tested as the patient seemed unable to move her eyes in any direction. A slight degree of external strabismus was noted in the left eye. Both pupils were widely dilated and reacted but sluggishly to light. The muscles of the lower jaw manifested a certain degree of tension but presented no paralysis. The right side of the face seemed somewhat flatter than the left. Deglutition was difficult and phonation impossible. The patient could not protrude her tongue or move it in any direction. There was no stiffness of the neck muscles, such as occurs in meningitis, but there appeared to be no power to move the head in any direction.

Examination of the blood and spinal fluid was negative on repeated tests. The blood count was normal and the blood culture sterile. Spinal fluid was withdrawn under a slight increase of tension and contained seven lymphocytes, no globulin and gave a negative Wassermann reaction. On several occasions, the urine showed a slight amount of pus but this promptly cleared up on proper treatment. In other respects, the urinalysis showed nothing pathological.

The patient has remained in this rigid, lethargic condition from February 2, 1919, until the present time. During this period the temperature averaged 102°. Once it became subnormal, and on the thirteenth day of the disease it suddenly rose to 106°, on the twenty-second and twenty-third

days respectively, to 105° and 104.5°, and on the thirtieth day, to 107.1°. Each elevation was followed by a sharp fall in the curve toward the normal line. The respiration and pulse did not tend to vary *pari passu* with the temperature; particularly was this true of the pulse, which showed a marked variability throughout the entire course of the disease.

The child is still alive and said to be improving slightly. At the present time she seems to have regained some power of expression and is able to smile faintly or indicate that she understands the questions addressed to her by gentle pressure of her hand.

These two cases are presented as typical of the cataleptic variety of the disease. One of them had previously suffered from influenza, while in the other no such history is obtainable. They are so strikingly alike in their principal clinical features as to justify the recognition of the cataleptic type of epidemic encephalitis.

III. PARALYSIS AGITANS TYPE

To this subgroup belong a number of cases characterized by the development of several of the more important symptoms of the Parkinsonian syndrome.

CASE VII. The patient, Mr. A. A., showed his first symptoms on January 21, 1919. At that time he complained of some headache, chilly sensations, and a nasal discharge. He felt ill but denied being nervous or depressed. His face and body were covered with perspiration at frequent intervals. His temperature was 100.2°. He had a few mucous râles in his lungs. On one examination his deep reflexes were exaggerated, and there was some muscular rigidity which had not been recognized by the patient. The diagnosis of influenza was made, and the patient received the influenza vaccine, remaining in bed until February 3d. At this time a lumbar puncture was made, 38 c.c. of fluid being withdrawn under greatly increased pressure. The Wassermann test of the fluid was nega-

tive, as was also the examination for tubercle bacilli. There were six cells in the fluid and the Noguchi test was negative; the sugar reduction was normal. On February 3d the patient became drowsy, arousing at times and occasionally getting out of bed. It was noticed that his extremities were stiff and trembling, especially on the right side, and the facial expression was fixed while the eyelids were kept closed. He gave the impression of great lassitude and lack of interest in his surroundings. Especially during the night there was a great deal of restlessness, which was not allayed by bromides given in considerable quantities. During the first two weeks in February, the patient at times was out of bed and saw a few friends, but he seemed incapable of any sustained interest. The stiffness of the right arm and hand increased materially. The lassitude continued and on several occasions there was retention of urine. This condition of affairs continued until February 21st, when the patient evidenced less and less interest in his surroundings and the restlessness increased at night to a low muttering delirium. During the latter part of February, the patient became much quieter, sleeping soundly most of the time but talking and muttering while asleep. Profuse sweating continued to appear over the head and limbs, while the tremor of the upper and lower extremities on the right side became more pronounced. On February 28th the patient lapsed into a state of unconsciousness with incontinence of urine. The pulse became much weaker and the respiration shallow and hurried. On March 1st he passed into coma and died on March 2d.

The temperature was, in general, low, varying from 99° to 100.4° with occasional elevations to 102° and 103°, until February 23d, when it began gradually to ascend and continued to do so progressively to the end, the final temperature being 106.2°. The pulse varied between 98 and 110 until February 23d, when it also ascended until February 27th, then becoming so variable in force that an accurate count could not be

made. The respiratory rate remained constantly around 24 until the 24th of February, when it began to increase rapidly. A record of the pulse, temperature and respiration is shown in chart II. A distinct cerebral tache developed after the fourth week of the patient's illness and during the fifth and sixth weeks there was a marked rash over the abdomen. The entire duration of the disease was forty days. Several repeated examinations of the blood and spinal fluid failed to reveal anything pathological.

The neurological examination of this patient made in the third week of his disease showed a man of middle age, lying supine in bed with eyes closed and responding indifferently to questions. His face had a definite wax-mask expression and in the right arm and leg there was a tremor which was continuous during rest, fairly large in its amplitude and relatively slow, resembling in all respects the tremor of paralysis agitans and having the further characteristic feature in the fact that it disappeared when the patient moved arm or leg. All of the normal automatic association movements of the face and eye, of eye muscles and neck muscles, of the intrasegmental movements of the arm and the intersegmental movements of the arm and leg were suppressed. It was possible to arouse him sufficiently to make tests of his gait and station. While he was rising from the bed the complete suppression of the normal automatic associated movements, especially upon the right side, was observed. In standing he had a typical Parkinsonian attitude and his gait presented a marked festination. When questioned, he complained of a feeling that made him fear that he might pitch forward when walking or standing and he vouchsafed the information that he felt that he could run better than he could walk. His whole attitude and appearance were unmistakably those of a Parkinsonian syndrome, more pronounced upon the right side of the body. On returning to bed, he immediately lapsed into a somnolent state

from which he could be readily aroused sufficiently to coöperate with intelligence. The reflexes of both upper and lower extremities were present and active, being slightly more active upon the right than the left side. There was no Babinski or other pathological reflex observed at any time during the course of his disease. The abdominal reflexes were present as were also the ciliospinals on both sides. The muscle tone in the right arm and leg was distinctly increased as compared to that upon the left side. Beside the tremor at rest, there were no other irregular, involuntary acts recorded.

The tests for somesthetic sensibility, in so far as the patient was able to coöperate, showed no departure from the normal, all qualities and modes of sensibility being intact.

Cranial Nerves.—The patient was able to recognize several different aromatics in either nostril. His vision was 20/20 in each eye without correction; the perimetric fields were impossible of definition on account of lack of coöperation; the pupillary reactions and the fundi were normal; a slight degree of nystagmus was present at times; there was no strabismus and no ptosis.

With the exceptions mentioned, the third, fourth and sixth cranial nerves were normal. The trigeminal nerve showed no disturbance in its motor or sensory divisions.

In the facial nerve, no definite paralysis was observed, although attention has already been called to the immobility of the face, giving rise to a typical wax-mask appearance.

The ninth, tenth, eleventh and twelfth nerves were all normal. There was, however, some distinct monotony in the voice such as is usual in cases of paralysis agitans.

This case of epidemic encephalitis has its chief interest perhaps in the fact that it presented the paralysis agitans type, rapidly running a fatal course

CASE VIII. Mrs. W. B., forty-nine years

of age, an active and robust woman who, in October, 1918, suffered from a severe attack of influenza and pneumonia particularly affecting the right lung. This condition kept her in bed for several weeks, and her convalescence was slow, so that she was not in a normal condition again until the beginning of December, 1918. For several weeks she went about as usual seeming to enjoy good health; but about the middle of December she was stricken again with an acute attack giving rise to fever, headache and general malaise. The exact nature of this disorder was not at the time clearly understood. Her temperature did not at any time exceed 103° , but continued for several weeks. Even after the temperature had ceased, however, she did not seem to regain her health; she suffered from a marked asthenia, not merely of physical strength but in initiative as well. Several days after the fever had left her she awakened one night to find herself in a profuse perspiration and her right arm shaking somewhat violently; and this tremor in the right arm and hand continued without cessation or much modification until the time of her death. She was unable to sleep during the remainder of the night, and upon arising in the morning noticed that a similar tremor was present in the right leg and foot. She was conscious that all her movements, particularly on the right side, had become annoyingly and perceptibly slower and that everything she did was, in her own words, "greatly slowed down." At the end of a week, her whole appearance and attitude had changed; the tremor was not the only feature of her trouble which annoyed her; for she felt a stiffening of the muscles in her arm and leg, together with a great sense of disquietude throughout her entire body. When she attempted to walk, she found that her gait was shuffling and slow. She herself made the observation that her speech had a mournful monotony about it for which she could not account.

Upon neurological examination at this time, she presented a typical Parkinsonian

attitude in standing as well as lying down. There was a tremor at rest in the right upper and lower extremities with marked hypertension in all of the muscles of the body. All automatic associated movements in the face and eye muscles, in the neck and arm muscles, in the arms and legs, were either abolished or distinctly decreased in their activity. The gait was shuffling, with some festination and a subjective sense of prostration. The patient stated that she felt she could run better than she could walk. One very striking feature of the case was the marked sense of disquiet and a desire to move the limbs from place to place but finding no position in which they were quite comfortable. Every position assumed very quickly lost the little relief which it originally afforded, so that the patient was in a state of constant mental and physical unrest which preyed upon her night and day, disturbed her sleep and harassed her when awake.

The deep reflexes of the right arm and leg were somewhat more active than the corresponding reflexes upon the left side. The abdominal reflexes were absent, due probably to the fact that the abdomen was greatly distended by a large cystic fibroid of the uterus. There was no Babinski on either side and no pathological reflex of any kind. The blood and urine examinations were negative. The spinal fluid was not examined.

At the end of two months, it became necessary to interfere surgically for a pelvic condition which seemed to jeopardize the patient's life. In consequence, a laparotomy was performed, the operation being a short one, lasting only one hour, gas, ether and oxygen being the anesthetic. The patient, however, never regained consciousness and died in thirty-six hours.

This case was the subject of several consultations and much difference of opinion. By some it was considered as a purely functional disorder, having the character of a hysterical tremor. This supposition was substantiated by the fact that the patient, prior to her illness, had been

engaged in war work among soldiers returning from France, many of whom were suffering from "shell-shock." Ultimately, however, the clinical features of the disease were too definite to leave room for doubt in the matter of its close correspondence to a typical Parkinsonian syndrome. The case is remarkable for the rapid development of its paralysis agitans features as well as for the absence of any conspicuous period of lethargy or somnolence.

CASE IX. Mr. B., forty-two years of age, married, merchant. The course of the disease in this patient stands out in pronounced contrast to the two cases already cited. Although a definite Parkinsonian syndrome developed, its full evolution was slow and its course short, the patient making a complete recovery.

During the summer, Mr. B. had complained of some intestinal disturbance, was predisposed to lassitude and some disinclination to participate in his usual occupation and amusements. In the early part of December, it was discovered that he was suffering from a tapeworm. Other than this, his previous history contained nothing of particular note as he had always been a man who enjoyed good health and was unusually active. He did not suffer from influenza so far as he and his family can recall.

In January, he went to a sanitarium to take vermifugic treatment, which proved successful in all respects. Several days after this treatment, instead of feeling improved, he complained that he was no better; in fact, he thought he felt much worse than before. He showed no disposition to get up and be about but preferred to remain in an inert if not semistuporous condition most of the time. He had no particular restlessness nor did he show any mental disturbance, but his conduct was marked by an unusual indifference to his surroundings. After remaining for one week in this condition, he began to see double, while his physician noticed that there was a slight palsy of the right side of the face. His indifference increased; he

seemed to have lost all initiative and nothing succeeded in gaining or holding his attention. He was neither stuporous nor comatose; when addressed he would respond in a listless manner.

He could be aroused and made to get out of bed. Upon such occasions his physician noticed that the attitude of his body, the position of his arms and head, the expression in his face, and his movements in walking were strikingly similar to the appearances of paralysis agitans without the tremor.

The patient was seen in consultation and the diagnosis of brain tumor was made.

At the end of four weeks, however, there appeared in the left foot a tremor typical of paralysis agitans. This slowly extended in the course of the next few days until it involved the entire left leg and finally manifested itself to a slight degree in the left hand and forearm.

Neurological examination at this time showed a patient with a characteristic wax-mask expression, in addition to which there was a slight facial palsy upon the right side and an occasional diplopia due undoubtedly to a weakness of the right oculomotor nerve. There was a slight degree of ptosis of both eyelids.

The tremor in the left foot and leg was unquestionably of the agitans type, not only in its rate and amplitude, but also because it occurred during rest and disappeared during action.

The reflexes on the two sides were active and equal. There were no pathological reflexes observed, either deep or superficial, although there was a slight tendency toward increase in tone of the muscles upon the left side. The patient had some festination in the gait and complained of a sense of procursion.

Sensibility was normal in all qualities, and the special senses, with the exceptions already noted, were intact.

The patient continued to be apathetic and generally somnolent for several weeks, but by degrees these symptoms disap-

peared, and at the end of two months not only the tremor and stiffness of the muscles but the Parkinsonian attitude and expression had entirely disappeared.

This patient did not have fever at any time during his disease.

His urine and spinal fluid were negative. Eventually he made what is considered a complete recovery and is now in a state of health which he himself believes to be normal.

CASE X. Miss H. M. H., thirty-eight years of age, office worker. Coincidentally with the onset of her last menstrual period, March 1, 1919, she suddenly became excessively nervous, could not sleep at night and was in a state of depression which made it impossible for her to attend to business. She believes her menstrual period was quite normal and that she did not suffer at that time or previously from illness which in any way resembled influenza. In fact, she is quite certain that she has not had the influenza during the past winter; and up to the time of her present illness she had been carrying on her work with her usual energy and application. Almost immediately upon the onset of her illness, she noticed a great disquietude, especially in her lower extremities. She found she could not get them in any comfortable position but was compelled to move them about continually from one position to another. She also noticed that her movements became much more slow and deliberate and that whereas she had always been alert and active, she now felt stiff and fixed as if molded into an attitude out of which it was difficult for her to extricate herself. She had a definite sense of procursion and thinks that she could run better than she walks. This proves to be the fact and when she starts running it is difficult for her to come to a stand. Her attitude and expression are typically Parkinsonian; after the first week of her present illness, she observed a tendency to a tremor appearing in her hand, and more noticeably in her left foot. This subsequently extended so that it involved the

entire left leg. The tremor came on especially when she was sitting down and when the leg was at rest. It was not noticeable while walking or standing. About this time there appeared over her entire body a rash which she said was not unlike hives.

A neurological examination made at the end of the fourth week of her illness showed a young woman looking and acting in all respects like a typical case of paralysis agitans. She sat immobile and then suddenly got up and walked about as if distressed, although her movements were slow. There was a suppression of almost all facial expression and of all automatic associated movements. When she got up or sat down it was almost as if her body were one piece. She walked slowly with no automatic associated movements in the arms. Tests of coordination and skilled acts were normal, save for a marked slowing down of all movements. Her voice was monotonous and toneless. When at rest there was a definite Parkinsonian tremor in the left leg and foot. The reflexes were normal. There was some increase in muscle tone, especially on the left side of the body.

All types of somesthetic sensibility were intact.

The Cranial Nerves.—The olfactory sense was normal. The vision seemed fair; the fields were normal. The fundi showed a slight blurring in the upper margin of the right disc. The pupils were unequal, the right being larger than the left. In size, they were round and regular and central in position. There was a slight hippus. Both pupils reacted to light and convergence, the left more actively than the right. There was no strabismus or other abnormal movements of the eye.

The acoustic nerve was normal and so also were the trigeminus, facial, glossopharyngeus and vagus, spinal accessory and hypoglossal nerves.

The patient's mental condition became so distressing that it was necessary to send her to a sanitarium. Here she had several severe hysterical attacks but subsequently

became calmer and has recently shown some slight improvement in all of her symptoms.

This case has its interest in the fact that the Parkinsonian syndrome developed apparently without any background of general systemic involvement, there being no history of temperature or other signs of acute infection. Furthermore, the absence of any pronounced lethargy and the apparent substitution for it of marked restlessness, insomnia and physical disquietude give a turn to the disease which is quite unusual when compared with the other cases of this type already reported.

CASE XI. Mr. G. G., age sixty, merchant.

During the winter of 1918-19, he had the influenza twice, the first attack of which was complicated by lobar pneumonia. He made a complete and good recovery after each attack and was gradually regaining his strength, when it was noticed that he began to lose his usual interest in his surroundings and lapsed into an apathetic condition. Every effort was made to arouse him from this state, but without success. He had no actual lethargy, but he slept much longer and apparently more deeply than was his custom. He would sit about the house most of the day without manifesting interest in his customary pursuits. All of his motor activities became decidedly slower and more deliberate. His gait was somewhat shuffling in character and he noticed a desire when walking to hasten his steps; in fact he felt like running most of the time and could actually run much better than he could walk. His wife and friends noticed a pronounced change in his expression. His face had a fixed look. His voice grew feeble and monotonous. At the end of a month, it was difficult for him to get about, not alone because his movements were slow, but also because of the great fatigue which any muscular exertion caused him.

Neurological examination at this time showed a well-preserved and robust man lying supine in bed, unable to rise without assistance, and quite as unable to turn

himself from side to side in bed. He complained bitterly of a sense of disquietude in his legs, and expressed the wish to move them frequently about in the bed. In this way only was he able to be comfortable.

His coordination was normal. There was no tremor in any part of his body. His reflexes were equal on the two sides, both deep and superficial, and no pathological reflexes were observed. Upon testing his muscles, the tone presented a marked increase in all of the muscles of both upper and lower extremities. All automatic associated movements were suppressed.

Somesthetic sensibility was normal.

Cranial Nerves.—The nerves of special sense were intact. There was a slight weakness and flattening of the right side of the face and the patient reported that in the early part of his illness he had suffered from a transitory diplopia.

The trigeminus, glossopharyngeus and vagus, spinal accessory and hypoglossal nerves were normal. On the day of the examination the patient manifested a tendency to sleep, which was more pronounced at this than at any other time during his illness. He had no temperature; the pulse and respiration were normal. At no time during his illness had there been fever or other evidence of systemic disturbance.

The laboratory findings were not reported in this case, and the patient has remained in practically the same condition for the past few weeks.

This group of cases has its chief interest in the fact that all developed clearly defined Parkinsonian symptoms. Four of these cases, in addition to the suppression of the automatic associated movements, the slowness of somatic movement, the characteristic attitude and facies, presented the agitated tremor.

One case, however, having all of the other cardinal symptoms, had no tremor.

Two of the five cases developed a moderate degree of somnolence during the course of the disease; on the other hand, three of

these patients showed a marked restlessness, not only at the beginning of the disease, but more or less throughout its entire course, a fact which makes them stand out in contrast to the other types of epidemic encephalitis already described.

One of these cases ran a fatal course after several weeks' duration, with a marked sustained elevation of temperature and a sharp rise during the few days preceding death.

One patient died due to an intercurrent operation.

One made a rapid, complete recovery, and two others are still suffering from the effects of the disease.

Three of the patients had suffered from an attack of influenza a short time before the onset of the encephalitis; two, however, did not give any such history.

IV. POLIOENCEPHALITIC TYPE

In this subgroup, of which many instances have been reported, the authors have encountered two cases. This group is recognized because the essential symptoms of the disease confine themselves almost wholly to the local results of the polioencephalitis, usually of a light grade, and causing a paralysis of one or more of the cranial nerves. The oculomotor (third nerve) and the facial nerve are most commonly affected, either in combination or separately. Lethargy, stupor or somnolence were not prominent in this group, although a pronounced asthenia of considerable duration was such a conspicuous clinical feature as to lead to the supposition that the cases might in reality be myasthenia gravis pseudoparalytica. Upon investigation, however, it was definitely shown that such was not the fact and that cases of this polioencephalitic type belonged, in all probability, to a mild or even abortive form of epidemic encephalitis.

CASE XIII. Mr. A. L. Age sixty-three, married, merchant. In January 1919, after many months of active and exacting busi-

ness engagements, the patient became so extremely exhausted that he consulted his physician. He was advised to give up business for a time and leave the city in order to rest and get the benefit of a complete change. The following morning his physician was again called, because the patient said he was suffering from some disturbance in speech which he thought to be aphasia and probably due to a stroke. He was particularly alarmed because on the previous evening when he had been at the theatre, he was struck by the fact that he was troubled with double vision. This diplopia persisted until the next day, although in the morning upon waking up he found he was quite free from it. It returned, however, after he had attempted to read the morning papers. At this time when seen by his physician it was noticed that he presented a bilateral facial paralysis involving all three branches of the facial nerve on both sides. This paralysis accounted for his difficulty in speech, for he talked as one would if a finger were pressed into either cheek. A slight degree of ptosis of both upper eyelids was also present. He was advised to leave the city at once for the seashore. This trip, however, was not successful, for on the second day his asthenia increased so much that he became alarmed and had what seems to have been a nervous chill, with acute indigestion.

Neurological examination at this time showed a man in a much enervated condition, fully cognizant of his environment, able to be up and about, and thoroughly to cooperate. His gait showed nothing peculiar, but his face had the characteristic expression of a bilateral facial paralysis. There was a marked epiphora of tears and a tendency to sialorrhea. His gait was normal. Muscular strength and coordination were good. There were no defects in skilled movements. His reflexes, deep and superficial, were normal and active. There were no abnormal, involuntary acts, such as tremors or twitchings, and all automatic associated movements were intact.

General sensibility was normal, as were

all of the cranial nerves with the exception of the seventh on both sides. The electrical reactions of the facial muscles, both direct and indirect, showed a fairly active response to the faradic current and the normal galvanic contraction formula.

The asthenia which was such a pronounced symptom in the early part of the disease has gradually disappeared. The facial palsy is slowly improving, although this has persisted for sixteen weeks.

CASE XIII. Mr. A. G. A., thirty-six years of age; clerk. During the time when a number of patients were confined in a suburban hospital suffering from the influenza, this patient was admitted because he had been slightly delirious and had had a rise of temperature to 102° which lasted for several days. Upon the disappearance of the acute symptoms he manifested a paralysis of both oculomotor nerves leading to a slight degree of ptosis and external strabismus. The right side of the face showed some flattening in its middle and lower portions, while the activity of the frontalis upon the right side was less vigorous than that upon the left. This patient developed no further symptoms, and a neurological examination made on the fourth day of his illness showed no evidence of pathological changes in his nervous system other than those which have already been noted. He suffered from an asthenia of a mild grade which lasted for about a month. His blood, spinal fluid and urine showed nothing abnormal. In six weeks he was sufficiently improved to enable him to return to his business, and since that time, with the exception of a brief attack of severe sciatica on the left side, he has enjoyed normal health.

Both of these cases may be taken to represent the strictly focal expression of a polioencephalitis of mild degree. The inflammation touched the nervous system but lightly and in restricted areas. In one case the involvement of the cranial nerve nuclei was of considerable severity and determined a slow convalescence; in the other instance, however, the affection of

the motor cells must have been slight, for the recovery was rapid and complete. These cases may, therefore, be taken to indicate the possibility of an abortive form of the disease, one in which the involvement is so light as to occasion a transitory clinical outline of the more severe type and to permit a *restitutio ad integram* almost before the symptoms were well enough defined to attract attention. In one case there was a general systemic reaction as shown by the slight rise in temperature; in the other case no such reaction occurred. It is of further interest to note that in neither case was it possible to obtain a history of influenza.

V. ACUTE ANTERIOR POLIOMYELITIC TYPE

CASE XIV. Over a year ago one of the authors* reported the case of a child 4 years of age whose mother stated that he had wasting of the right leg, dragging of the right foot, and weakness of the right leg, particularly when walking or running, and especially when walking any distance. This child's trouble occurred during the last epidemic of poliomyelitis which swept over New York City in 1916. The child left New York for Liverpool in September, 1916. On the last days of his trip he complained of feeling ill and his body seemed hot. He was seen by an English physician, who made the diagnosis of infantile paralysis, upon hearing that the child had come from New York. On the second day of his illness, the patient passed into a deep sleep, in which condition he remained, not even arousing to take food, for the next fourteen days. The mother took the child to the hospital and placed him upon a bed on his right side. Each time she called to see him he was still in the same position. After fourteen days the boy made his first sound, calling his mother. The child was unable to hold up his head without assistance for six months. He sat up alone

* Poliomyelitis with Prolonged Somnolence. Frederick Tilney, M.D. Neurological Bulletin, Vol. I, No. 1, January, 1918, page 7.

for the first time in February, 1917, and two weeks later he was able to stand. Since that time he has gradually improved, the right leg remaining much smaller and softer than the left. With the exception of considerable atrophy and a marked hypotonus of the right lower extremity, the neurological examination made in 1918 showed nothing abnormal in the patient. There is no record of the patient's hospital findings, so it is impossible to state whether the child suffered from an ophthalmoplegia or whether he had any cranial nerve involvement. At the time the case was considered to be one of acute poliomyelitis, and this opinion was undoubtedly greatly influenced by the fact that the illness occurred during a widespread and virulent epidemic of this disease, during which clinical imagination was, perhaps, too keenly alert in the recognition of the disorder then epidemic.

This condition of prolonged somnolence associated with a lower motor neuron type of paralysis affecting the leg, created much discussion, although it was the final opinion of the physicians who had an opportunity to study the case that the diagnosis was an atypical poliomyelitis. In no other way did it seem possible to explain the unusual and prolonged somnolence. It is our present belief that the case does in reality belong to that latter group and presents that rare but not at all impossible combination of symptoms, namely, the simultaneous involvement of brain and spinal cord. It is noteworthy, however, that in the majority of cases of encephalitis, the spinal cord is but little involved, and conversely, by far the greater proportion of acute poliomyelitis cases shows little or no involvement of the brain stem. Particular emphasis is laid upon this fact, and the authors desire to call special attention to this case of infantile paralysis which occurred during the height of a virulent epidemic of acute anterior poliomyelitis and yet had one of the most prominent symptoms associated with epidemic encephalitis, i.e., prolonged somnolence.

VI. POSTERIOR POLIOMYELITIC TYPE

So far as the recorded cases go at the present time, this type presents an unique variety of the disease whose chief characteristic is an inflammatory affection involving the dorsal root ganglia, in other words, producing an acute posterior poliomyelitis. The authors have encountered but a single instance of this type.

CASE XV. Miss M. S. Age thirty-nine years. The patient's previous history is not important in her present condition. She has suffered from no serious illness, and so far as she is able to state, did not have influenza during the past winter. Her family history is negative.

On January 1, 1919, the patient attempted in the morning to arise from bed, but upon standing up, fell to her knees, and only with great effort was she able to return to bed. This incident did not seem to have alarmed her much or to have attracted her attention especially. She knew that she felt weak and sleepy and so decided to remain quiet. In a short time she fell asleep and for the next three weeks, she slept most of the time. She could be aroused to take her meals but manifested little interest in her surroundings. When seen by her physician, her condition was thought to be one of a nervous breakdown, possibly an hysteria. Her temperature was not taken and it is believed that she had no elevation of temperature during her acute illness. As she began to recover at the end of three weeks, she noticed, upon attempting to read, that the letters blurred, and when looking at a distance, she was conscious of double vision. This, however, rapidly disappeared, so that in the course of a week she could read for a short space of time without seeing double; but as soon as she became at all fatigued, the double vision returned. She also noticed a tingling and burning sensation which was very disagreeable and at times painful upon the left side of her face in an area which came exactly up to the midline and followed in all its boundaries the cutaneous innerva-

tion of the trigeminal nerve. In the region in which she suffered this painful sensation, there appeared a number of small herpetic-like eruptions which, however, never assumed the full character of herpes; these small eruptions were very sensitive to the touch, to the air and to cold water. At the end of ten weeks the rash was still faintly visible and was discretely limited to the area of innervation upon the skin of the left trigeminal nerve. About this time the patient began to complain of a tingling and burning sensation on the dorsal aspect of the right leg, corresponding to sacral dermatomes 2, 3 and 4. This she described as similar in all respects to the sensation in the left side of her face, although no eruption had as yet been detected in this area on the right leg.

The neurological examination showed that the reflexes were equal and active upon the two sides, both deep and superficial. The muscle tone was normal. There was no paralysis or loss of strength and no incoördination. The muscular status was normal.

Somesthetic sensibility, with the exception of the left trigeminal area and the regions of innervation corresponding to sacral dermatomes 2, 3 and 4 upon the right leg, was normal. In addition to the pain and dysesthesia described in the two affected cutaneous zones, there was a marked hyperalgesia in the trigeminal area of the left side and the cutaneous zones on the back of the right leg. Paresthesia was also present in these regions, both cold and heat being interpreted as pain, and a slight stroking with cotton wool giving a distressing sensation.

This case, whose cardinal symptoms were lethargy, ophthalmoplegia and zoster-like involvement of the left side of the face and the right leg, may be regarded as a typical example of acute encephalitis. Its unusual clinical feature arises from the fact that the dorsal root ganglia, certainly the Gasserian upon the left side and probably the dorsal root ganglia of sacral 2, 3 and 4 upon the right side, had been involved in an inflammatory process.

VII. EPILEPTO-MANIACAL TYPE

CASE XVI. Mr. G. H., thirty-six years; clerk. In the latter part of December, 1918, this patient suffered from influenza with severe bronchial involvement. His recovery was unsatisfactory, for the reason that he seemed to pass from a respiratory to a psychic disturbance. In the second week of his illness, he became unmanageable. His temperature still remained at 102° and 103°. He had some hallucinosis as well as certain ill-defined, unorganized delusions. These were more troublesome at night, for during the day he slept most of the time. For several weeks he lay in this lethargic condition, occasionally during the day muttering some incoherent words or showing a partial coöperation when aroused to take medication or food. At this time he was orientated to time, place and person. At the end of two weeks his somnolence during the day became more profound, while at night his delirium would rise at times to a point at which he became violent and destructive, so that actual restraint was required. During these periods of excitement, he manifested definite hallucinosis and delusions, the latter being mostly of the persecutory form. On several occasions he managed to free himself from his restraint and presented such maniacal manifestations that it was necessary to overcome him by force and keep him under the influence of narcotics. In the fifth week of his illness, he had a generalized, epileptiform convulsion, which was repeated in several hours, and from that time until his death, these convulsions continued with increasing frequency and severity. His temperature slowly rose and after being in a status epilepticus for several hours, he died in coma after an illness lasting six weeks.

Neurological examination of this patient showed nothing of localizing significance. The motor system in so far as it could be adequately investigated, was normal, except for a slight external strabismus of the right eye, together with some nystagmoid movements affecting both eyes.

The blood count and culture were negative on several tests. The first lumbar puncture showed a spinal fluid under considerable pressure containing a slight increase in cells. Upon smear and culture it revealed the presence of an organism which was regarded as a diplostreptococcus. This finding, however, should be accepted with much reservation, since the spinal fluid withdrawn several days later and tested in three different laboratories, gave no such results. No autopsy was obtained.

VIII. ACUTE PSYCHOTIC TYPE

CASE XVII. F. S., male, age thirty-six. Admitted to the hospital October 19, 1918, with a diagnosis of influenza and bronchial pneumonia, and the final diagnosis of acute encephalitis and acute parotid sialadenitis.

On October 15, 1918, the patient was seized with a chill and general malaise. He felt some weakness and had headache and fever with pain in the back. On examination, the patient presented much prostration, flushed face and rapid breathing; he had a coated tongue and a red pharynx. There were signs of broncho-pneumonia in the lower right lobe posteriorly. There was some abdominal distension.

Neurological examination showed a patient, very apathetic, lethargic and difficult to arouse. There was nothing abnormal in the cranial nerves. The patient seemed unable to protrude the tongue.

Examination of the blood made on October 21st, showed the white cells 7,200; polymorphonuclears 96 per cent; large mononuclears 2 per cent and lymphocytes 2 per cent. The patient slept all of the time and was too somnolent to cooperate in any examination. There was some evidence of parotitis. He could be aroused from his lethargy with difficulty but when he responded at all his answers and actions were irrational. During the next week his apathy and somnolence increased steadily, although there were intervals, especially

at night, when he gave evidence of an acute psychosis. He evidently suffered from auditory hallucinosis, as he seemed to hear voices and reply to them. Consciousness was much clouded and the patient became entirely disorientated during such intervals as when he was aroused to take food. During the night he manifested a vicious tendency to resist all efforts to care for him. He would get out of bed, urinate on the floor, and insist on throwing the bedclothes on the floor or out of the window. If left to himself, he would wander about the ward. Ultimately he became noisy and uncontrollable.

On the 28th of October, the lethargy had deepened into a stupor. A lumbar puncture made at this time showed a clear fluid under no increase of pressure, with 4 lymphocytes, globulin reaction negative, Wassermann reaction negative, culture sterile.

It now seemed impossible for him to articulate or to move his tongue and swallow. The reflexes in the upper extremities were present but sluggish. The abdominal reflexes were all present but sluggish. The knee-jerks were present but feeble, the right being slightly more active than the left. There was no Babinski or any modification of this sign. The pupils were dilated, regular and equal, but reacted sluggishly to light, and accommodation. The optic discs were sharply outlined. His temperature is shown in Chart III.

On October 30th the patient passed into coma, and on November 1st died. No autopsy was obtained.

IX. INFANTILE TYPE (NEONATORUM)

It seems fitting in this subgroup to mention the cases of three infants, respectively four, four and six weeks of age; all of these cases were seen at the Babies' Hospital through the courtesy of Doctors Robbins and Goldbloom, who are preparing a carefully detailed report for later publication. In the clinical picture,

these patients corresponded so exactly to the conditions seen in adults suffering from acute encephalitis, that there can be little doubt as to the pathology of their disease.

The interest of the cases lies in the fact that their disease began at such an extreme early age, an observation, so far as the writers know, to which attention has not been hitherto called.

CASE XVIII. This infant, six weeks old, without premonitory symptoms, suddenly refused to take nourishment and passed into a deep sleep in which he remained for two weeks.

During this period it was necessary to feed him by tube. Due to the excellent care which he received, he was kept alive, but when he began to show a return of interest, it was found that he had a complete paralysis of the left side of the face. This paralysis has persisted for a number of weeks although it is slowly improving.

CASE XIX. This patient, four weeks old, gave a similar history to that of the one already described and after a period of three weeks' somnolence, began to recover. In this instance, the lower and middle facial muscles upon the left side were found to be paralyzed as a result of the disease. The child is now doing well and is gradually improving.

CASE XX. Another infant, four weeks old, had a similar history of prolonged somnolence for two weeks, at the end of which time it began to show some return of interest. Subsequently it was found to be suffering from a paralysis of the left side of the face and a motor paralysis of the right musculo-spiral nerve, giving rise to a characteristic wrist drop upon that side.

These three cases are cited in brief abstract only as they will be described later in *extenso* by the pathological department of the Babies' Hospital. The writers desire to express their deep appreciation of the courtesy and kindness of the staff of the Babies' Hospital which made possible the observation of these three cases.

DISCUSSION

Critical Analysis of the Symptoms and Course of the Disease in the Series of Cases Described.

Onset.—In general it may be said that the onset of epidemic encephalitis is sudden and usually occurs without the development of prodromata. Eighteen of the twenty cases described in this article showed such a sudden onset, while in the remaining two, there was a short prodromal period characterized by general malaise, asthenia, slight chilliness and a rise of temperature. This lasted for several days before the definite symptoms of the disease were recognized.

2. *Lethargy.*—Profound somnolence approaching stupor was the most common clinical feature in the group of cases described. This lethargy was characterized by the fact that although the patients seemed to be sleeping soundly and to be oblivious to the incidence of ordinary stimuli, they could be aroused by more active stimulation and would then, for a short time, manifest a fairly clear orientation and show little or no clouding of consciousness. This alert state, however, was short lived and as soon as the effort to keep the patients awake ceased, they would lapse back into the deep sleep again. The lethargy was of varying degrees of intensity; in some instances it was extremely light, being little more than a continuous drowsiness from which the patients could be aroused without much difficulty. In other cases, however, the somnolence was much more profound and required an active effort to awaken the patients. Seventeen of the cases suffered from this lethargy, while three of them, although typical of the disease in other respects, at no time showed an inclination to somnolence or drowsiness. It is interesting to note in this connection that all three of these cases belong to the type described in the subgroup of paralysis agitans.

3. *Cranial Nerve Palsies.*—A large percentage of the cases showed involvement of

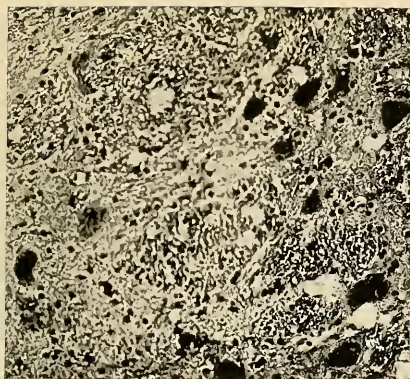


FIG. 1. OCULOMOTOR NUCLEUS.



FIG. 2. HEMORRHAGE IN MEDULLA.

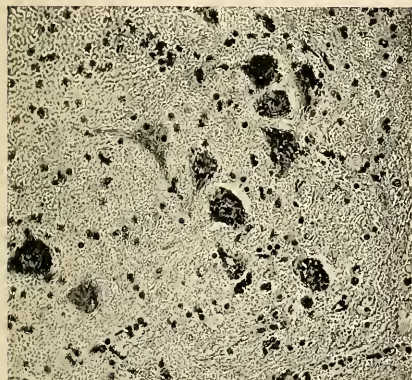


FIG. 3. HYPOGLOSSAL NUCLEUS.

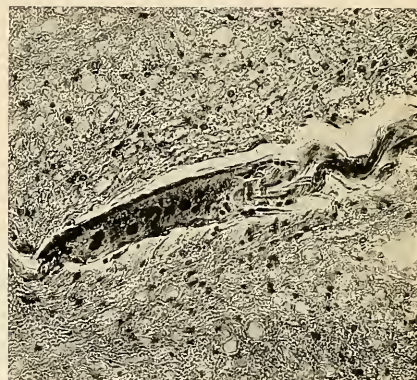


FIG. 4. VASCULAR INFILTRATION.

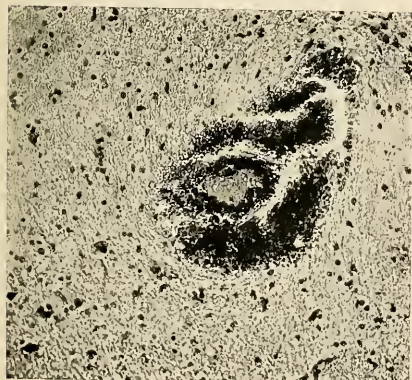


FIG. 5. HEMORRHAGE IN THE THALAMUS.

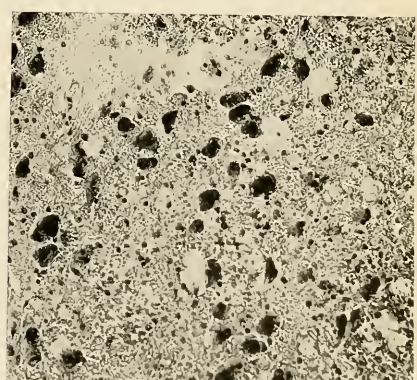


FIG. 6. ABDUCENS NUCLEUS.

one or more of the cranial nerves, fourteen of the cases presenting such affections. Of these, seven were confined exclusively to the oculomotorius nerve (the third nerve). Six cases presented a combination of the oculomotorius and facial nerve palsies, and in one case, which rapidly ran a fatal course, the third, sixth, ninth, tenth and twelfth cranial nerves were simultaneously involved. In all instances, the cranial nerve palsies were of the lower motor neuron type. Six cases of this series presented no cranial nerve involvement.

4. *Temperature*.—Some degree of pyrexia was the rule. It is, however, not an essential feature of the disease. Numerous observers have reported cases in which no temperature has been observed throughout the entire course of the illness. On the other hand, it is quite possible that some degree of fever of short duration may be present in all cases but escapes detection, in much the same way as in poliomyelitis, of which there are cases reported without elevation of temperature. This may be due to the fact that such cases did not come under observation at a time when the temperature might be accurately determined. It seems probable that all cases, even of the milder grades, are subject to changes in temperature at some time during the course of the disease. Of the twenty cases of this series, fourteen ran a sustained temperature between 101° and 102° , with several instances showing acute exacerbations much above this level for a number of days and, indeed, a few of them for a number of weeks. In the lethargic type a temperature averaging 102° seems to be the rule. There may, moreover, be sudden elevations to a much higher level (105° , 106° or even 107°), the temperature range having the appearance already shown in Charts I, II and III.

The acute psychotic and epilepto-manic types show a tendency to a decided rise in temperature which is maintained for a considerable time.

Of the five cases of paralysis agitans type, one alone showed a distinct and pro-

longed elevation of temperature. This proved to be a fatal case.

In the milder cases of the lethargic type, the elevation of temperature is usually of short duration. In the more severe cases, however, the temperature curve may run a course coincident with the lethargy and begin to show an inclination to return to normal only as the patient recovers from the somnolence.

In those types of the disease in which the inflammatory process is confined to the brain stem, the temperature does not seem to reach an unusually high level, even in fatal cases, while in the posterior poliomyelitic types the temperature rise may be so evanescent as to escape clinical record.

Such cases of the infantile type as have been observed would lead to the supposition that the pyrexia is not pronounced and is usually coincident with the duration of the somnolence.

The pyrexia seems to vary directly with the intensity of the infection, both as to the degree of its elevation and its duration.

5. *Tremor*.—Seven of the twenty cases presented a distinct tremor. In five of these, the tremor in its rate, amplitude and general conditions was of the paralysis agitans type. In two cases, on the other hand, it was of a much finer variety and made itself apparent either when the patient attempted to move a part or limb, or when the same was passively moved. The striking appearance of certain of the other cardinal features of the Parkinsonian syndrome in conjunction with the tremor aided in establishing the identity of the tremor itself. The wax-mask expression, the suppression of automatic associated acts, the Parkinsonian attitude, the monotony of the voice, the flexible hypertonus of the muscles, the shuffling and festinating gait, were so unmistakable as to leave no doubt with reference to the clinical picture. In every instance, the distribution was that of a hemi-tremor, in two cases involving the right side and in two others the left side of the body. It was a tremor of

Temperature, Pulse and Respiration Charts in Three Fatal Cases of Epidemic Encephalitis

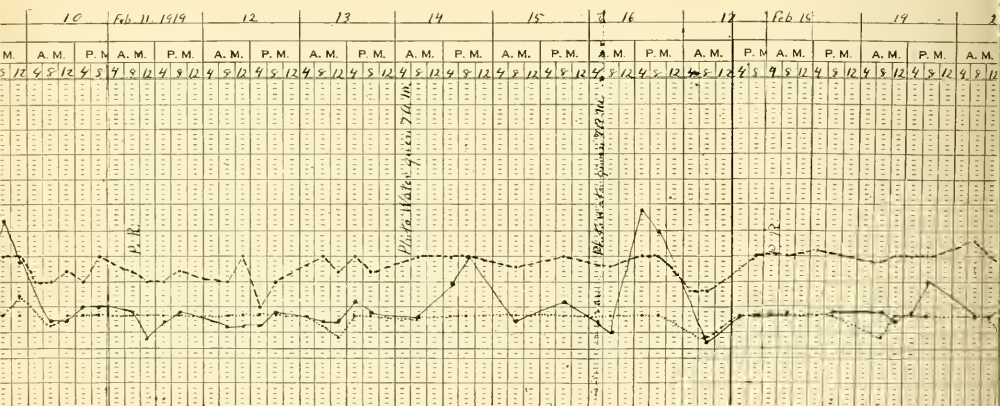
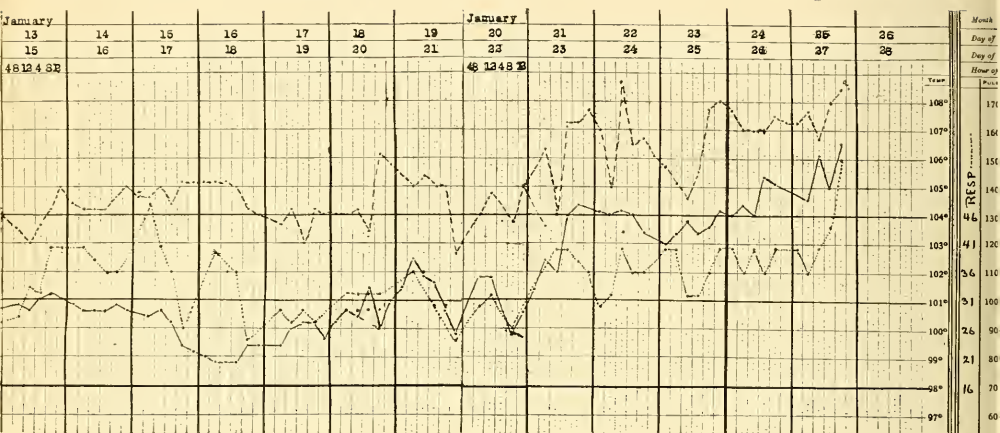


CHART II. PARALYSIS AGITANS TYPE

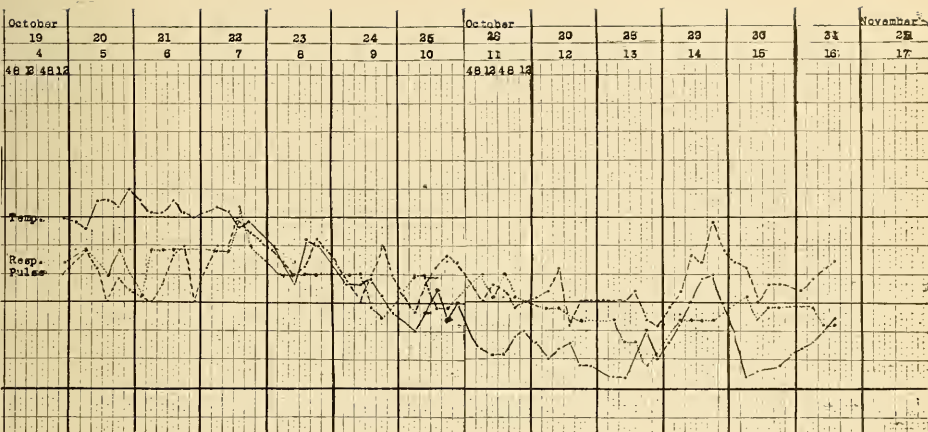
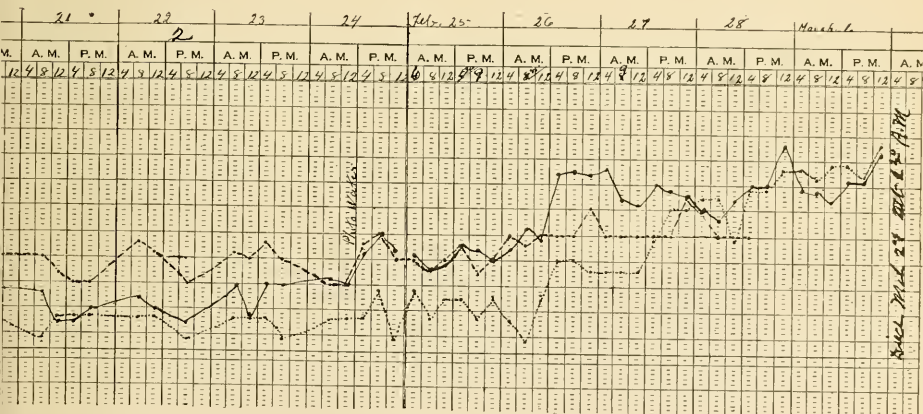


CHART III. ACUTE PSYCHOTIC TYPE



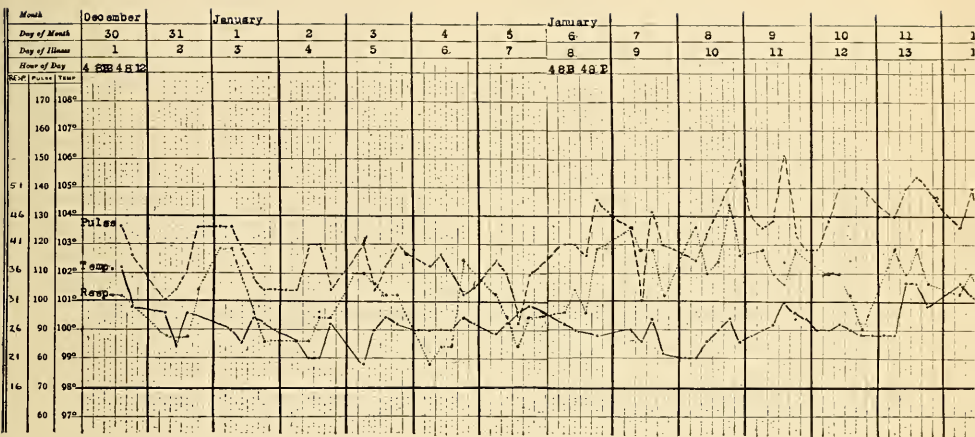
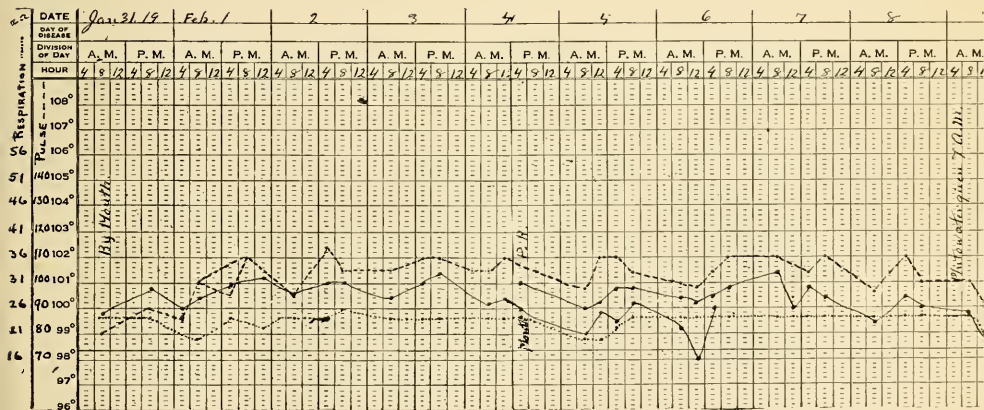


CHART I. CATALEPTIC TYPE



Temperature, Pulse and Respiration Charts in Three Fatal Cases of Epidemic Encephalitis

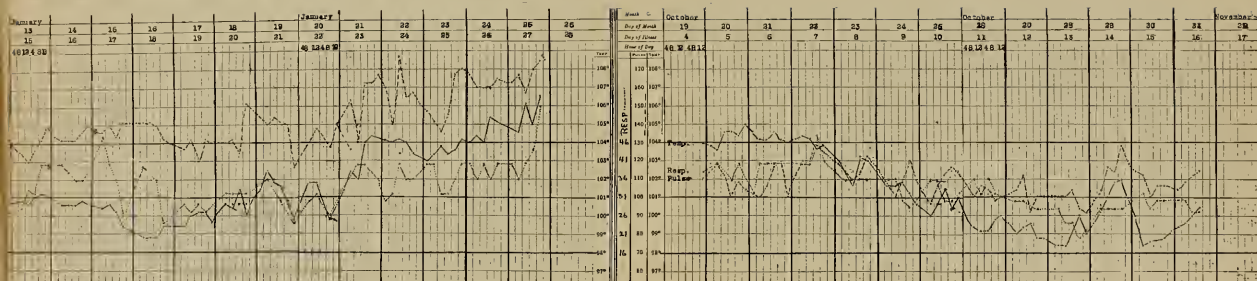


CHART III. ACUTE PSYCHOTIC TYPE

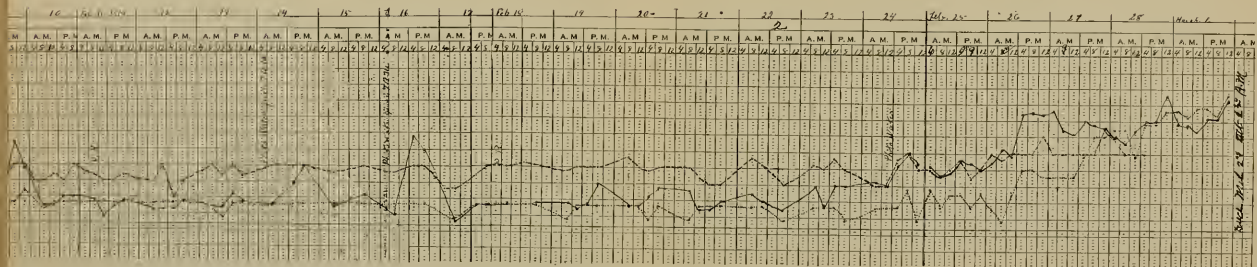


CHART II. PARALYSIS AGITANS TYPE

rest which usually disappeared when the patient was in deep sleep but in nearly every case would make its reappearance when the patient was sleeping lightly or was about to awaken. One case had all of the characteristics of a paralysis agitans *sine agitatione*. Such tremor, therefore, as was noted, belonged for the most part to the paralysis agitans type, although a finer and more rapid oscillation should also be included in the description under this heading.

6. *Asthenia*.—More or less profound and general asthenia was observed in six of the twenty cases; in fourteen, however, it was not a prominent symptom. In those instances in which it did occur, it was conspicuous for its long duration and also for the tendency for the muscular weakness with a sense of general debility to return upon slight exertion, even weeks after the acute symptoms had disappeared. Some of these patients, when well along in their convalescence, have found it necessary to go to bed again for several days at a time because their usual activities completely exhausted them.

7. *Restlessness*.—As if replacing the lethargy, restlessness and a sense of disquietude, which led the patients to feel that it was impossible to get the body or limbs into any comfortable position, especially while at rest, was a symptom which occurred in five of the twenty cases. It is of interest to note that this restlessness and sense of disquietude were present in all but one of the five cases belonging to the paralysis agitans group, and the fifth case showing marked restlessness was that of a patient whose subsequent history seems to indicate that he is developing some of the features of the Parkinsonian syndrome.

8. *Catalepsy*.—An unusual hypertonus in the muscular system determining fixed positions of the trunk and limbs, as well as of the facial and lingual muscles, was a striking feature in two of the cases. This hypertonus produced a rigidity which had some of the elements of a *flexibilitas cerea*.

On the other hand, the total absence of any evidence indicating implication of the pyramidal system would seem to place the responsibility of this muscular change outside of the palliospinal connection. Indeed, this symptom has been accredited to some extrapyramidal system. The hypertonus so generally seen in the paralysis agitans group of this disease undoubtedly has its explanation in the disturbance of the extrapyramidal connections. Furthermore, a slight but still discernible degree of hypertonus was observed in all of the lethargic cases, which might be looked upon as a partial involvement of the extrapyramidal fibers.

9. *Spinal Fluid*.—The spinal fluid was carefully examined in fifteen of the twenty cases. In only one of these was there anything observed which could be considered as possibly pathological. This occurred in the single case in which on one lumbar puncture a diplostreptococcus was found in the fluid upon incubation, on culture and in the stained smear. Subsequent tests of the fluid obtained on another lumbar puncture from the same case showed no such organism to be present. With this doubtful exception, the spinal fluid in all cases was sterile; it contained but a slight increase in globulin and there was no marked pleocytosis. In some of these facts, the laboratory findings in epidemic encephalitis stand out in contrast to the familiar features present in acute anterior poliomyelitis. Although no definite organism has as yet been found in the spinal fluid of the latter disease, the marked pleocytosis and increase in globulin have been taken as significant of meningeal irritation or inflammation.

COURSE AND DURATION OF THE DISEASE

The average duration of the disease in so far as it may be estimated at present, is about eight weeks. This figure, of course, included a number of cases which are still improving or have as yet shown little or no change for the

better. Some cases, on the other hand, may terminate fatally after running a brief course of a few days or as long as nine weeks. Many cases with the severest symptoms clear up rapidly. There is, however, a tendency to retrogression even after the lapse of several weeks. A few patients have shown a tendency for their symptoms to reappear and in one or two cases new symptoms have become prominent after the patient had made an apparently complete recovery.

Of the twenty cases included in this report, 25 per cent terminated fatally.

These figures seem to represent a mean between the mortality rate of Economo² which was 54 per cent, that of Netter⁸ which was 46 per cent, that of Wilson¹⁰ which was 15 per cent, and that of Hall³ who lost none of his sixteen cases.

Of the cases of this series which have survived, 15 per cent show a complete recovery, 20 per cent are improving slowly, and 35 per cent are either at present stationary or show some aggravation of their former symptoms.

PROGNOSIS

Conservatism should be used in giving a prognosis in cases of any type of this disease. Some forms of it seem to be more prone to fatal termination than others. In general, it may be said that the poliomyelitic and mild polioencephalitic types are not serious with respect to life and usually terminate in a complete recovery after a relatively short duration. Cranial nerve palsies, however, especially where the facial nerve is involved, are somewhat slower in repair than is the case with the ordinary peripheral facial palsies.

Ophthalmoplegias are generally of short duration but tend to reassert themselves for some time after recovery has been established, especially when the patient becomes exhausted or fatigued.

In the epileptomaniacal and acute psychotic types, the condition should be re-

garded as grave and not of hopeful prognosis as to life.

In the lethargic type, one should be governed largely by the intensity of the somnolence. This, however, is not the only index which may serve as a guide. Many of the most severe cases of this type make a good recovery and they should be considered as particularly grave only when the cranial nerves of the bulb give evidence of involvement.

The prognosis in the cataleptic type is much better than the general impression which these patients actually convey during the period of their illness. Their appearance seems to certify a fatal termination and yet a number of these cases make a good recovery. In our series, one case of the cataleptic type is still living and one of them died.

In the paralysis agitans type of the disease, it is perhaps too early at this time to state definitely what outcome may be expected in every case. It is quite certain that complete recovery from this type of involvement is possible, but whether all of the cases manifesting the Parkinsonian syndrome will eventually clear up is a question to which the material of this study cannot as yet give satisfactory answer. A number of other observers, however, have reported cases of the paralysis agitans type in which complete recovery has eventually occurred.

The infantile type of the disease (Neonatorum) seems to afford a fair outlook as far as the acute stage of the disease is concerned. Such patients as have been under observation survived the acute stage of the disease. On the other hand, all of these infants are still in a precarious condition since they suffer from the effects of a greatly impaired central nervous system which imposes a handicap upon them in meeting their somatic and visceral requirements.

During the inception of the acute stage of all cases of epidemic encephalitis a guarded prognosis seems to be indicated.

Other Types of Epidemic Encephalitis.—

A review of the literature makes it possible to recognize types of this disease other than those which have come under the observation of the writers, and while it is not yet possible to include these types in our series, they seem so well established in the experience of others, as to necessitate their inclusion here for the sake of completeness.

Among these are types which have been described as meningitic, polyneuritic, cerebellar ataxic, hemiplegic, diplegic and monoplegic. These varieties, together with those already mentioned in the series of cases under consideration, call attention to the protean character of the symptoms, a fact which accentuates the probability that no portion of the nervous system is exempt from the pathological changes of the disease.

DIFFERENTIAL DIAGNOSIS

The diagnosis of epidemic encephalitis is not without its difficulties. One of the writers was recently called to see a case of so-called "Sleeping Sickness" which had all of the clinical features of a typical case of epidemic encephalitis, even including the negative findings of the cerebrospinal fluid. Upon post mortem, however, the case was found to be one of tuberculous meningitis.

In another instance, a case which had acquired considerable reputation through the lay press as *Sleeping Sickness*, because of a trance-like state which had persisted for a number of weeks, finally showed a positive Wassermann reaction in the spinal fluid, although this test had on several previous occasions been reported negative.

Under all circumstances, laboratory findings must be evaluated with much caution and always with particular reference to the known reliability of the laboratory from which the returns are received.

Three classes of disease will offer some perplexity in the differential diagnosis.

The first group consists of diseases in which the diagnosis is made by examination of the spinal fluid.

1. *Tuberculous meningitis* differs from

epidemic encephalitis in that the spinal fluid is usually under increased tension, presents a pleocytosis and contains the tubercle bacillus.

2. *Cerebrospinal Meningitis* of the epidemic type differs from epidemic encephalitis in the fact that the spinal fluid is cloudy or flocculent, contains many leucocytes, in some of which may be recognized the pathogenic organism of the disease, the meningococcus.

3. *Cerebrospinal Syphilis* differs from epidemic encephalitis by the positive Wassermann reaction in the spinal fluid, a marked pleocytosis, increased globulin, and either a paretic or a luetic colloidal gold curve.

The second group of diseases consists of those whose recognition will be aided by certain pathological tests.

1. *Typhoid Fever*, the nature of which will be recognized by the Widal reaction, the temperature curve, the enlarged spleen and the rose spots.

2. *Diphtheritic Paralysis*, whose chief differential feature is the discovery of the Klebs-Loeffler bacillus in the throat and a history of suggestive diphtheritic infection.

3. *Uremia*, in which the routine tests of the urine show an albuminuria or the presence of renal casts. These findings may be substantiated by an estimation of the blood urea or of the renal efficiency by the phenolsulphonophthalein test.

4. *Botulism*.—This condition is distinguished by the failure to isolate the bacillus botulinus in the food partaken by the patient immediately prior to the onset of the disease and by the acute gastrointestinal disturbances.

The third group represents conditions which may be distinguished by their clinical features:

1. *Hysteria* is recognized chiefly by the presence of hysterical stigmata, the absence of pyrexia and the presence of marked sensory disorders, such as hemianesthesia or a contraction of the visual fields. The presence of normal reflexes and the absence of true palsies, nystagmus and

other oculomotor disturbances are also significant.

2. *Cerebral tumor*, a diagnosis made in one of the twenty cases of the present series, may be differentiated by the absence of optic atrophy, papilledema, vomiting and paroxysmal headache.

3. *Acute dementia* is distinguished by the persistence of maniacal delirium unaccompanied by stupor or pyrexia.

NATURE OF THE DISEASE

Whether this is a new disease or a recent outcropping of an old epidemic tendency is a much debated subject. The occurrence of prolonged and profound sleep in connection with epidemic diseases is not new in medical history. Such somnolence has been described in connection with many epidemics of influenza since the earliest times. Zuelzer¹² reports the fact that in an epidemic of influenza occurring in 1712, profound sleep was so frequent and pronounced a symptom that in Tübingen the disease came to be known as the *Sleeping Sickness*. In more recent times, Longuet⁴ in 1892 gives an account of a mysterious disease known as "*Nona*" also "*Nonna*". This was said to have occurred especially in northern Italy and Hungary and to be characterized by lethargy and weakness. The derivation of the word "*Nona*" seems to be obscure; the name was given to an alleged new form of disease reported in 1890 in northern Italy, Bavaria and Russia. It appears, however, that there is little foundation for the belief that there is such a new disease, since the reports seem to be founded on cases of typhoid fever with coma or of somnolence following influenza as well as of smallpox of an irregular and severe form. A recent epidemic of lethargic encephalitis in Vienna was described by Economo² in 1917 while Netter⁸ reported a similar outbreak in Paris in 1918. The report of an inquiry into an obscure disease, *Encephalitis Lethargica*, made by a com-

mission in London, in 1918, records the occurrence of 168 cases, together with a valuable analysis of this material. Wilson¹⁰, Hall⁸ and others have also made records of a number of cases of this disease during the same period in London.

In all probability, this is not a new disease, but an epidemic whose presence has attracted more wide-spread recognition and more careful analysis than in the past.

Epidemiologically, it must be regarded as an infectious disease, probably communicable. This, at least, is the attitude taken at present by the English authorities, for the disease has been made temporarily a notifiable one in England and Wales for the period of one year.

The bacteriological evidence thus far adduced does not throw much light upon the pathogenesis of epidemic encephalitis. Von Wiesner¹¹, studying one of Economo's cases, isolated a gram positive diplo-streptococcus in 1917. This he was able to cultivate and the culture of it injected in a macacus rhesus produced somnolence with muscular weakness, while peritoneal injection into guinea pigs caused death from internal hemorrhage. A filtrate from the original brain-cord emulsion of the patient through a Berkfeldt filter injected into a macacus rhesus produced no symptoms, although an unfiltered emulsion of the brain and spinal cord injected subdurally into the same species caused death in forty-six hours, the animal having previously presented profound stupor in addition to a paresis of the right hind leg.

McIntosh⁶ examined two cases and found in one a gram positive motile non-proteolytic anaërobic bacillus which resembled the *vibron septique* of Pasteur. In the other case, there was a gram positive non-motile anaërobic bacillus which belonged to the bacillus Welchii group of anaërobies. He found no bacillus botulinus and no pleomorphic coccus such as described by Rosenow in acute anterior poliomyelitis. McIntosh injected emulsions of the basal ganglia, pons, medulla and cervical cord intracerebrally and intraperi-

toneally in macacus rhesus but produced no results. In this respect, his findings are in direct contradiction to those of von Wiesner.

Cultures from the blood and spinal fluid were sterile, as were also those taken from the excreta of the naso-pharynx and from the feces.

Sera of recovered patients injected into mice did not protect these animals against injections of botulinus toxin. MacNalty⁶ suggests that epidemiologically, clinically, and pathologically, epidemic encephalitis differs from classical poliomyelitis and is, in fact, a disease *sui generis*. MacNalty suggests that the relation of epidemic encephalitis to poliomyelitis may possibly be that of typhoid fever to paratyphoid.

The pathological study of a number of cases establishes the fact that the tissue reaction due to the infection is an actual inflammation. Marinesco⁷ has pointed out that epidemic encephalitis may be differentiated from the changes of botulism and the alcoholic polioencephalitis of Wernicke by the fact that in these latter diseases the changes are not those of a true inflammation but rather due to profound alterations in the vessel walls, leading to secondary degenerative processes in the nerve tissue. He also calls attention to the fact that although the true sleeping sickness may resemble epidemic encephalitis in many respects, its differentiation depends upon discovery of the trypanosoma gambiense.

Marinesco and McIntosh both allude to what they deem essential pathological differences between acute anterior poliomyelitis and epidemic encephalitis.

In poliomyelitis, for instance, the distribution of the lesion is largely confined to the cord. The pia-arachnoid, as a rule, shows considerable involvement, while the changes in the brain are slight.

In encephalitis, however, the greatest changes are in the basal ganglia and nuclei of the brain stem, the gray matter of the floor of the fourth ventricle and around the aqueduct of Sylvius. The pia-arachnoid is

but little involved and changes in the spinal cord are rare.

The character of the cellular infiltration in the two diseases is also a distinguishing feature. In poliomyelitis, polymorphonuclear leucocytes predominate and plasma cells are of less frequent occurrence. Hemorrhages are common and relatively large while the vascular changes affect the smaller arteries.

In encephalitis, plasma cells are much more common while the polymorphonuclear cells are rare. Hemorrhages when present are usually microscopic. The infiltration is adventitial about the small vessels and more especially the venules.

The cellular changes in the two diseases are somewhat different. In poliomyelitis, degenerations are pronounced and neurophagia is active, while in encephalitis, the cells are not much involved; such changes as do occur are relatively mild and neuronophagia when present is not marked.

The clinical differences between poliomyelitis and epidemic encephalitis have been emphasized by Wilson and MacNalty.

It seems justifiable to conclude, therefore, from anatomical, clinical, pathological and bacteriological evidence, that these two diseases are not identical, although they may be closely allied in their pathogenesis.

Draper¹ thinks epidemic encephalitis is strikingly like poliomyelitis in many respects, and may be a subgroup of the latter. He is of the opinion that animal experimentation alone, carried on upon a large scale, can give the solution to this problem.

Much has been written concerning the relation of epidemic encephalitis to influenza. Many hints from the past and not a few confident assertions in the present aim to give these two diseases a single identity. There seems, however, little real basis for this position; but the question probably cannot be definitely decided until more is known concerning the exact pathogenesis of each of these clinical conditions.

In the series of cases described by the writers, fifty per cent of the patients gave no history indicative in any way of influenza, unless it be conceded that the abrupt onset of cerebral symptoms without the occurrence of any of the more usual symptoms of influenza were an expression of this latter disease. It is not unlikely that the relation between these two diseases may be a mutualistic symbiosis, or that one may be metabiotic to the other.

TREATMENT

No specific agent or remedy has yet been discovered in the way of serum or vaccine for dealing directly with the disease or affording immunity against it.

During the acute stage in all types of epidemic encephalitis, the patient should be kept in bed and, since this is probably a communicable disease, some degree of isolation should be established. Perhaps the barrier system or one in which nurse and patient are kept apart from the rest of the household, will suffice. All bedding, cloths, utensils, etc., which come in direct contact with the patient, should be properly sterilized.

The principle of treatment is essentially symptomatic.

For pyrexia, cold sponges or a light cold pack when the fever is 102 or over usually give the best results.

Urotropine, 10 grains three times a day, is theoretically presumed to be of benefit and may be used in this amount or in increasing doses.

Elimination is an important feature in the treatment; the bowels and kidneys should be kept active and flushed out as much as possible. For this latter purpose, high colonic irrigations of normal salt solutions should be given daily or if this proves fatiguing to the patient, two or three times a week. As much water as possible should be administered by mouth, and when this becomes difficult, the continuous Murphy drip is advisable.

Feeding should be careful throughout

this period and limited to a fluid diet. Frequent feedings of two to four ounces every two hours are preferable to larger quantities at longer intervals. Unless otherwise indicated, the diet need not carry more than a medium calorie value and only in cases where emaciation is marked and rapid is forced feeding necessary.

Repeated lumbar puncture has been advantageous in some cases, 10 to 20 c.c. of the fluid being withdrawn every three to five days.

In the more profoundly lethargic and cataleptic cases, care should be taken of the skin to prevent decubitus, and when indicated, cardiac stimulants, such as digipuratum and digalen, should be administered.

Restlessness is frequently a difficult complication. This seems to be best treated by means of the bromides, the usual combination of ammonium and sodium of potassium giving good results, in doses of 10 grains each, well diluted, three times a day.

For insomnia, 10 grains of medinol dissolved in warm milk, or 3 grains of allyl barbituric acid similarly administered at bedtime, usually produce sleep. For some patients, however, other similar remedies may be required, since those mentioned may not be as effective as certain other soporifics.

For active or violent delirium, the most efficient remedy is paraldehyde in doses of 4 drams every four hours if indicated. In certain cases, however, as for example those in which the maniacal tendency is most developed, the prolonged warm bath gives excellent results. This, however, should not be attempted by inexperienced attendants and should be under the direct supervision of the attending physician. In cases showing violence or active delirium, restraint should be avoided as much as possible. It is much less exhausting to the patient if he be permitted to carry out, in part at least, the more or less indefinite purposes arising from the activity due to his delirium. If circumstances will permit, it is better not to attempt to restrain the

patient in bed, but allow him some latitude, while surrounding him with the necessary protection against injuring himself or others.

For the tremor, either of the agitans type or the more rapid oscillation, a prescription containing hyoscine hydrobromate, gr. 1/150 with codeine sulphate $\frac{1}{4}$ gr. may be given three times a day. For these cases, morphine is best avoided, and the bromides, if used at all, should be administered in small doses, that is, not exceeding 30 gr. a day.

During the stage of convalescence, care should be exercised in not permitting the patient to get up and about too early. This error frequently leads to relapses and unnecessarily prolongs the asthenia consequent upon the disease. A partial rest-cure plan should be adopted when the patient is first attempting to get about. This should never be left to the discretion of the patient, but prescribed in detail, according to the hours of rest required, the amount of exercise or engagement in usual occupation and such other directions as seem necessary. During this time a simple nutritious diet and plenty of water are essential.

For the asthenia seen so frequently in these patients, an excellent remedy is suprarenal residue, 5 minims, three times a day after each meal. This may be best administered by mouth in combination with the hypodermic injection of cacody-

late strychnia, gr. 1/60 every day either with or without the arsenate of iron.

During the stage of aftercare of the palsy, especially the facial paralysis, much benefit may be had from daily facial massage and the application of the galvanic sinusoidal current in mild strength directly over the facial nerve for fifteen minutes three times a week. The faradic current is of little use, especially in the early stages of the paralysis, when faradic response is generally absent.

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FEVER IN HYSTERIA, WITH THE REPORT OF TWO CASES

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The occurrence of pyrexia with hysteria is of rather infrequent occurrence and has always been of considerable interest because of the difficulty in determining whether the alteration in temperature was of hysterical origin or due to some other complicating condition. In the observation of two cases about to be described in which hyperthermia and hysteria were present, there were noted certain features which it is believed are characteristic of the fever of hysteria and may be of value in differential diagnosis between the fever of infectious and that of hysterical origin.

CASE I

Patient: F. McG., female, aged twenty-one, single, born in England. Admitted to Presbyterian Hospital August 25, 1915.

HISTORY. Chief Complaint.—Vomiting, headache, fever and abdominal pains.

Family History.—Patient's own father died while the patient was a baby; cause unknown. Her stepfather died three years ago of tuberculosis. The patient was living in the same house with him. Her mother is alive but has heart trouble. One sister died of tuberculosis at six years of age. Two sisters and three brothers died in infancy. There has been no trouble in any other member of the family similar to the patient's complaint.

Personal History.—Patient has worked for three months as a ward maid in the New

York Orthopædic Hospital. Before that she was employed as a maid at the City Hospital. She has been in this country for three years.

Her appetite is fair, bowels regular. She drinks very little water; two cups of coffee a day; alcohol denied. She has no frequency of urination; no nocturia; no pain. Menstruation began at eighteen, always regular at intervals of four weeks. No dysmenorrhœa; no leucorrhœa. Her average weight is 127 pounds.

Past History.—Measles at two years of age and again at six years. Scarlet fever at four; whooping cough at eight; mumps at twelve. No diphtheria, pneumonia, rheumatic fever or typhoid fever. Both knees were broken after being run over in the street, at the age of seven. She was in bed four months at this time but the fractures healed perfectly. She had an attack of appendicitis six months ago; the appendix was removed at the City Hospital. Shortly after this she had swelling of the gums, treated with boiling water under ether at the City Hospital. She has had no previous attack similar to the present illness.

Present Illness.—Headache and loss of appetite for two weeks before the present illness began. The onset was sudden, nine days ago, when she awoke early in the morning with nausea. She vomited and brought up undigested food and a lot of green, sour-smelling vomitus. At the same time she had a sharp, severe pain in

the middle of the epigastrium which was well localized. Vomiting relieved the pain on the first day. On the second day she began to have slight pain in the left upper quadrant just under the costal border and also around her old appendix scar. The vomiting continued up to the day before admission. She would vomit every time she attempted to take anything by mouth, even medicine. The vomitus at no time contained blood, but consisted of an immediate return of whatever she had eaten. On the day before admission she managed to retain a little broth, but was unable to retain anything else. For six days she has had a daily gastric lavage, and ever since starting this the pains around the appendix scar and in the left upper quadrant have been worse. The bowels have not moved regularly since the present illness began, but she has been having enemas every two days. She does not have much gas in the stomach but everything seems to sour immediately. She has not been jaundiced. She has had severe throbbing frontal headache ever since the onset of the present illness. For the past four days she has been unable to void and has been catheterized every few hours. She has had no chills but has had fever ranging from 99° to 102° ever since the illness started. She is very dizzy. On attempting to sit up two days ago, her vision was quite blurred, but it is normal now. She has had no cough. Since the onset she has not slept well.

The mother states that the patient was a normal child in every particular. She never had convulsions, enuresis, or sleep-walking, nor did she cry out in her sleep. She was rather precocious, and walked at eight months. She was normal in her mental development and was considered bright at school. She had no paralyses. She does not know of any great mental shocks except the accident in which her limbs were broken at the age of seven. The mother had seven miscarriages before this daughter was born.

Physical Examination.—The general ap-

pearance is that of a well nourished, well developed white female, lying quietly in bed without dyspnoea, cyanosis or pain, but with moderate prostration. She is mentally clear and intelligent.

Skin.—There is a large cherry-red nevus occupying almost the entire right side of the face, extending a short distance into the scalp and down to the upper lip and along the mucosa of the lip down on to the upper gum to the teeth margin, the soft tissue about the teeth sockets being heaped up in this region and almost burying the teeth. There is an extensive folliculitis and scattered acne pustules over the entire chest, back and abdomen. The skin is quite dirty, rough and moist. There is no jaundice or cyanosis.

Superficial Lymph Nodes are not pathologically enlarged.

Head is normal in size and shape, and is symmetrical. *Eyes:* conjunctivæ clear; scleræ white; pupils medium size, round, clear, regular, centrally placed. React to light and on accommodation. No ocular palsies; no nystagmus. *Ears and Nose:* No discharge or abnormality to external examination. *Mouth:* Teeth very irregularly situated, many are carious and there is a moderate pyorrhœa. The tongue is protruded in the midline without tremor. The tonsils are small. There is no exudate or sign of inflammation. Pharynx is clear.

Neck.—No stiffness, rigidity or tenderness. No pathological pulsations; no thyroid enlargement.

Thorax.—Symmetrical; expansion good and equal on the two sides.

Lungs.—Resonant. Voice and breath sounds all normal. No râles.

Heart.—Maximum impulse in the fifth left space, 9 cm. from the midline. The sounds are clear and of good quality. No murmurs, thrills or reduplications. Action regular. Pulse equal, regular, synchronous, well sustained with moderate force and tension. Arterial wall not palpated. Blood pressure 112-70.

Abdomen.—Rounded, plump and soft. No tenderness, masses, or free fluid. There

is a three-inch scar resulting from the appendix operation in the right lower quadrant. There is considerable tenderness in the region of the scar and in the left upper quadrant just below the costal arch, but no muscular resistance and no mass can be made out.

Liver.—Dullness at fourth space, flatness from the sixth to the costal margin; edge not felt.

Kidneys and Spleen not felt.

Extremities.—No paralyses, atrophies or deformities. No edema; knee jerks present and active; no pathological reflexes.

Rectal Examination.—No masses—entirely negative.

Ophthalmoscopic Examination.—The disks are normal except that at the nasal side the disk of the right eye is a little hazy. The vessels are normal. There are no hemorrhages.

LABORATORY EXAMINATIONS.

8/25/15. *Urine Analysis*:

Specific gravity	1.026
Reaction	Acid
Color	Amber
Turbidity	Slight
Precipitate	Flocculent
Albumin	Very faint trace
Glucose	None
Acetone	None
Diacetic acid	None
Microscopical examination	Few epithelial cells

8/25/15. *Blood Culture*: Negative.

8/25/15. *Blood Wassermann*:

Alcohol antigen	Negative
Cholesterin antigen	Negative

8/26/15. *X-ray of Colon* shows no evidence of obstruction or other abnormality.

8/26/15. *Blood Count*:

Hemoglobin	75%
White blood cells	9,200
Polymorphonuclear leucocytes	75%
Lymphocytes	24%
Eosinophiles	1%
No malarial organism found	

8/27/15. *Spinal Fluid*:

Amount	9 c.c.
Pressure	Normal
Appearance	Clear and colorless
Coagulum	None
Sediment	None
Total leucocytes	15
Red blood cells	None
Globulin	None
Culture	Negative
Wassermann	Negative to both alcohol and cholesterin antigens

8/27/15. *X-ray of Chest* shows nothing abnormal.

8/27/15. *Blood Count*:

White blood cells	8,400
Polymorphonuclear leucocytes	68%
Lymphocytes	31%
Eosinophiles	1%
Malarial organisms	None

8/28/15. *Von Pirquet Reaction*: Suggestive.

Morro: Negative.

8/28/15. *Blood Count*:

White blood cells	9,000
Polymorphonuclear leucocytes	70%
Lymphocytes	27%
Eosinophiles	3%

8/30/15. *Blood for Widal*: Negative.

8/30/15. *Blood Count*:

White blood cells	10,200
Polymorphonuclear leucocytes	72%
Lymphocytes	28%

8/31/15. *Blood Smear*: Malaria parasites not found.

9/1/15. *Culture from Urethral Smear*: Few pus cells; no gonococci.

9/2/15. *Blood Wassermann*: Negative.

9/4/15. *Blood Count*:

White blood cells	4,000
Polymorphonuclear leucocytes	77%
Lymphocytes	21%
Basophiles	2%

9/4/15. *Examination of stool* shows no blood, pus, ova or parasites.

9/9/15. *Blood Count*:

White blood cells	7,800
Polymorphonuclear leucocytes	76%
Lymphocytes	20%
Eosinophiles	4%

CLINICAL NOTES:

8/31/15. Patient's symptoms since admission have been very much as she described them in the last few days before admission. She has had occasional vomiting spells and very severe headaches, some abdominal pain and a great deal of tenderness, most marked since admission in the right lower quadrant, and a fluctuating temperature ranging from 99° to 103°. The lungs have been perfectly clear; no indications of meningeal disturbance have been present.

Vaginal examination has not been attempted, due to an imperforate hymen.

Rectal examination (by Dr. Hawkes) showed some general induration but no masses or tenderness. He does not advise any kind of operation.

It has been necessary to catheterize her about every twelve hours and large amounts of urine are obtained each time. Since admission there has been no voluntary micturition.

Rectal examination was made again today (8/31/15) by Dr. Hawkes who thinks there may possibly be a focus of irritation or infection around the base of the old appendix stump, but that in view of the fact that she does not seem to be suffering from sepsis it would be better to continue waiting than to operate now.

9/1/15. It is reported at the City Hospital that the appendix was freely movable, had a fecal concretion in the middle of it and at the tip. Diagnosis: Catarrhal inflammation. Microscopical examination not made.

9/2/15. The tenderness in the left upper quadrant has entirely disappeared, but the tenderness around the old appendix scar seems to be more acute. There is absolutely no muscular rigidity. Catheterization continues to be necessary and large amounts of urine are frequently obtained. She vomited again last night and felt somewhat relieved after it. She continues to have rather severe headaches.

9/3/15. Last night and this evening the patient has shown very peculiar spells of

unconsciousness. She had a premonition tonight that an attack was coming, saying she felt nervous and sick at her stomach. About five minutes afterwards, while talking to the nurse, her head fell suddenly to one side, her arms dropped and she became unconscious. At the onset of the attack there were a few involuntary twitchings of the jaw but her mouth was not closed tightly. She then lay quietly on her back with the chin up and the arms thrown out at each side, absolutely unconscious and shaking violently. Sticking her with a needle and pressing deeply over the tender area in the right lower quadrant failed to elicit the slightest response. There was no rigidity of the neck or any portion of the body. The color was good, the heart was regular and not rapid. The rate was about 80; the pulse was quite strong and regular. Blood pressure 116-64. The hands were almost ice cold and rather clammy, but the feet were warm. Respirations were somewhat irregular—one deep respiration would be followed by a period of about ten seconds of apnoea; then another similar deep respiration followed possibly by one very shallow respiratory movement. The total respirations were only about six to eight per minute. The jaw was closed pretty tightly but not immovable. On placing the flat of the hand over the front of the chin, a fine tremor of the jaw could be distinctly felt. There was no frothing at the mouth and the tongue was not bitten. The eyelids were closed and on opening them the eyeballs showed a tremor and both eyes failed to coördinate perfectly. The pupillary reflexes were normal; knee jerks were present and active. There was no Babinski.

After the patient had been in this state of total unconsciousness for about fifteen minutes, she commenced to move her extremities, then a sighing which developed into a groaning, the patient saying repeatedly, "Oh, my head!" and then consciousness gradually returned so that she answered questions perfectly. She complained of severe pain in the frontal region.

After a conscious interval of about five minutes she fell into unconsciousness again, and had a repetition of the attack described above, for about three minutes. The performance was repeated again a third time, the unconscious period lasting about five minutes, and on awakening the patient complained of severe pain in the head. During this third attack the respirations were distinctly changed, being very shallow and rapid, about 48 to 54 per

and commenced to feel faint and sick at her stomach. Then she became dizzy, everything whirled around before her, and then she fell asleep. She remembered events earlier in the evening perfectly, but nothing at all during the time of the three attacks. Her complaint now is quite moderate, but she still asks that something be given to relieve the pain which she says runs from the frontal region of her head to the occiput. She is mentally clear

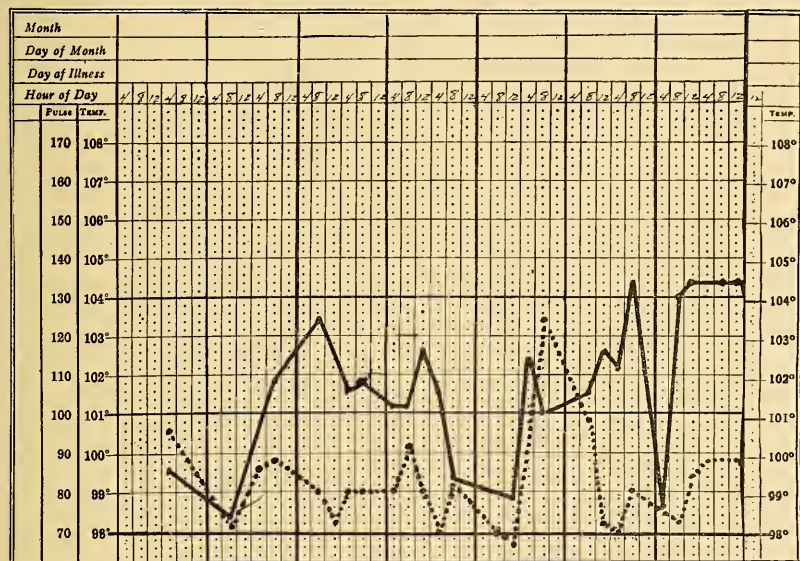


FIG. 1. CHART OF TEMPERATURE AND PULSE CURVES FOR A PERIOD OF ONE WEEK SHOWING THE DISSOCIATION OF THESE PHENOMENA. TEMPERATURE ——— CONTINUOUS LINE. PULSE DOTTED LINE.

minute. The pulse remained slow and strong, rate 72. The temperature during the third spell was 98.3°.

Fifteen minutes after the last attack the patient was completely herself again, sitting up in bed; she asked that the restraining sheet be taken off, and when told that she had just been having a spell, she laughed and apparently would not believe it. She stated that just after catheterization she lay down on her back

and bright. Her hands are still rather cold; the pulse and respirations are normal.

9/4/15. This morning the patient has no recollection of what happened during the night, but volunteered the information to one of the nurses that she had had a similar "spell" in the Orthopaedic Hospital on August 16, just after a lavage. She now complains of headache and pain around the appendix region.

9/6/15. The tenderness in the region

of the appendix scar is very much less this morning than heretofore. For the past two nights the patient has been in an unconscious state most of the night.

Ear Examination.—Both drums are normal. Landmarks and cone of light distinct. No bulging or redness.

10/2/15. On September 30, as the patient still continued to be quite noisy and troublesome at night, it was decided to transfer her to Bellevue Hospital. However, she refused to go, as she said she was afraid of the place. She was told that she would be given one more trial, but if she had the slightest attack of unconsciousness or became noisy, she would be transferred without delay; also that she would not be catheterized, and if she did not void she would be transferred. The result was almost magical, and since then she has voided regularly, and has had no attacks of unconsciousness or shouting. She still has moderate tenderness in the right lower quadrant. The temperature still ranges between 100° and 103°.

10/9/15. The patient had another attack of unconsciousness and was transferred to Bellevue.

During her stay in the Presbyterian Hospital the patient was on a soft diet. She was in the Hospital from August 25 to October 9. Her weight during this time was as follows:

August 26	127 pounds
August 30	129½ pounds
September 6	128½ pounds
September 13	129 pounds
September 15	129 pounds
September 21	126 pounds
September 27	126 pounds
October 4	124 pounds

Her respirations were constantly from 18 to 22.

CASE II

PATIENT: J. C., female, aged sixteen, single, born in New York. Admitted to the Presbyterian Hospital, March 7, 1919.

HISTORY. *Chief Complaint* is fever and pain over the right cheek for one and a

half years; attacks of asthma for fourteen years.

Family History.—Father and mother alive and well. Two sisters and four brothers all alive and well. No chronic diseases, mental or nervous disturbances. Mother has had no miscarriages.

Past History.—Broncho-pneumonia at two years of age; has been subject to chronic bronchitis ever since. Measles, whooping cough in childhood. Typical asthmatic attacks began three years ago. All other illnesses denied.

Personal History.—Appetite good. Bowels regular. Average weight about 110 pounds, which has not changed since the onset of the illness. Does not use tea or coffee. Urination once or twice a day. No nocturia. Sleeps poorly. Menstruation began at sixteen. Her periods are regular, each one lasting four days; no dysmenorrhœa.

History of Present Illness from St. Luke's Hospital records.—In December, 1917, on account of asthma, she had a submucous nasal operation. Ten days later her right ear drum was incised. In January, 1918, the ethmoid cells were opened and drained. In March, 1918, she was admitted to the Manhattan Eye and Ear Hospital and her right antrum was opened and drained. In May, 1918, she was admitted to St. Luke's Hospital for attacks of asthma and tenderness and pain in the right cheek.

On admission to St. Luke's Hospital the patient was markedly dyspnoeic. Her right cheek was very tender; all over her chest there were loud sibilant and sonorous râles. Her heart showed an accentuated pulmonic second sound, but was normal in outline. The abdomen was distended. The temperature was 100.2°. The red blood count was 3,800,000, with 70 per cent hemoglobin. The white blood cells were 12,000, polymorphonuclear leucocytes 76 per cent, lymphocytes 20 per cent, eosinophiles 4 per cent.

A foreign body was removed from the right ear and the patient was discharged.

On May 30, 1918, the patient was admitted again to St. Luke's Hospital

with a temperature of 103° . Diagnosis, bronchopneumonia or miliary tuberculosis. The spinal fluid was negative; the sputum showed no tubercle bacilli, and no other evidence of tuberculosis was found. The patient was discharged and referred to the Manhattan Eye and Ear Hospital for treatments of her antrum.

On September 23, 1918, the patient was again admitted to St. Luke's Hospital with a temperature of 105° . As the general physical examination was negative, except for the fever and tenderness over the right cheek, the patient was transferred to the surgical side and the antrum again

noisy and very emotional. X-ray of the intestines showed some redundancy of the pelvic colon with ptosis of the cecum and transverse colon. X-ray of the chest showed a slight infiltration of the right upper lobe. X-ray of the teeth was negative. On account of the abdominal distension and possible signs of fluid in the abdomen, she was thought to have tuberculous peritonitis or an ovarian cyst. A laparotomy was performed but nothing abnormal was found except that the intestines were markedly distended with gas. She was discharged from the hospital February 17, 1919, with the cause of the temperature

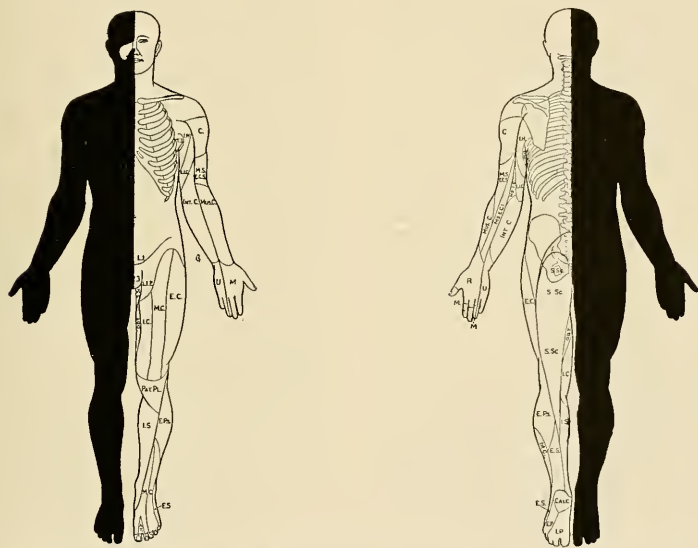


FIG. 2. DIAGRAM SHOWING AREA OF ANESTHESIA TO TOUCH, PAIN, TEMPERATURE, POSTURAL AND VIBRATORY SENSE.

explored, but nothing abnormal was found. X-rays of her chest were negative. Retention of urine was noted from October 26 to December 1, when she was discharged in good physical condition, but still with a temperature ranging from 100° to 103° .

She was again admitted in January, 1919, with a temperature of 105° being

unknown and with a diagnosis of essential fever.

On March 7th she was admitted to the Presbyterian Hospital where this summary of the present illness was obtained.

Summary of Present History.—She has always been subject to bronchitis since she had pneumonia at the age of two.

Typical asthmatic attacks began three years ago and she has had several since. Otherwise she felt very well until a year and a half ago when she noticed pain in the region of the right cheek; she also had fever. She consulted a nose and throat specialist in one of the dispensaries and had a submucous nasal operation, then a myringotomy on the right side, and a month later an operation on the ethmoid cells. Two months after onset she went to the Manhattan Eye and Ear Hospital

sisting. Later it was thought she had tuberculous peritonitis and a laparotomy was performed, but a normal abdomen was found. Two months ago she was discharged from St. Luke's, no diagnosis having been made, still suffering from fever which is continuously about 101° to 104° . There have been occasional asthmatic attacks and constant pain and tenderness in the right cheek. She has had no chills or sweats. She has had occasional attacks of vomiting. The appetite is good,



FIG. 3. LATERAL VIEW SHOWING POSTURAL SPINAL CURVATURE.

where her right antrum was opened and treated for several months without improvement. She was then referred to St. Luke's Hospital, where she was admitted about seven months ago. Shortly after admission she contracted influenzal pneumonia and upon recovery had two operations upon the right maxillary sinus. No improvement followed, temperature and tenderness of the right cheek per-



FIG. 4. LATERAL VIEW SHOWING WEAKNESS OF RIGHT ARM AND LEG. THE APPARENT NORMAL STATE OF NUTRITION IS ALSO EVIDENT IN SPITE OF A TEMPERATURE RANGING FROM 98° TO 104° FOR ONE AND ONE-HALF YEARS.

the pulse regular, she has had no cough and no loss in weight.

Her physical examination on admission to the Presbyterian Hospital, on March 7, 1919, was as follows:

A young, white female, aged sixteen years, lying propped up in bed in acute distress. She is vomiting and greatly ex-

cited, dyspnoëic and flushed, but does not seem to be in pain. Mentally she is clear but too excited to answer questions. Her head is symmetrical, there is no evident abnormality. The skin is soft and smooth; the surface temperature is elevated. The face is quite flushed. The neck is not stiff

and regular; react to light and on accommodation.

Nose.—Slight mucopurulent discharge from the right nostril.

Ears.—No tenderness or discharge.

Mouth.—Teeth in good condition except two which are slightly carious. Tongue

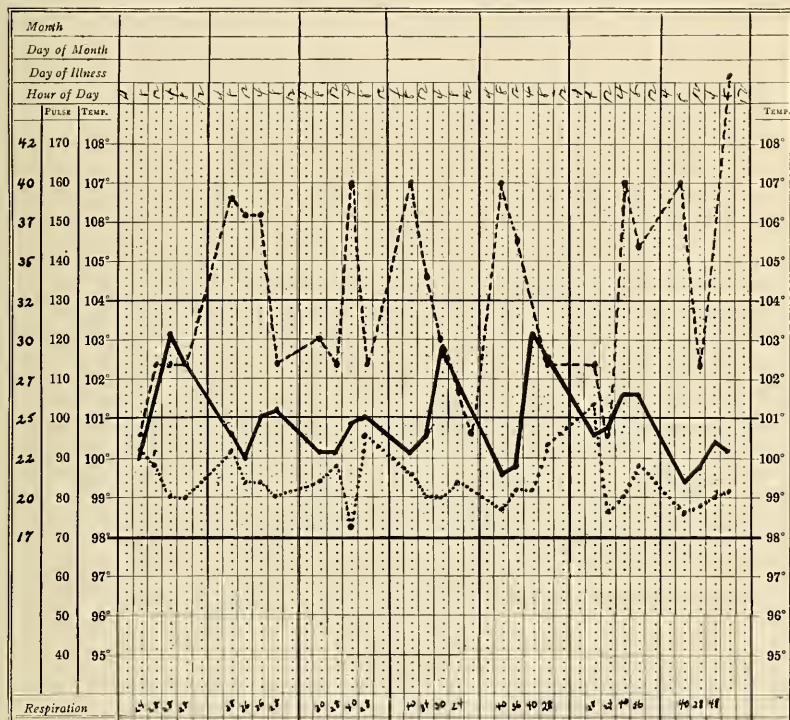


FIG. 5. CHART OF TEMPERATURE, PULSE AND RESPIRATION CURVES FOR A PERIOD OF ONE WEEK SHOWING THE DISSOCIATION OF THESE THREE PHENOMENA.

TEMPERATURE — CONTINUOUS LINE.
PULSE DOTTED LINE.
RESPIRATION ----- INTERRUPTED LINE.

or tender. The thyroid is not enlarged and there are no visible pulsations.

Face.—There is marked tenderness over the right maxillary sinus; no swelling or redness.

Eyes.—Conjunctiva slightly congested; movements normal; pupils circular, equal

slightly coated. Tonsils not enlarged or inflamed.

Thorax symmetrical, well developed.

Breasts normal.

Lungs, hyperresonant throughout. There are everywhere many sibilant and sonorous râles, and expiration is much prolonged

and labored. The accessory muscles of respiration stand out prominently.

Heart is not enlarged or displaced; the left limit of cardiac dullness is $9\frac{1}{2}$ centimetres from the midline in the fifth space. The sounds are distinct and of normal character. There is a slight systolic murmur heard in the pulmonic area. The pulse rate is 120. It is regular, medium size, with a normal pulse wave. The peripheral vessels are soft.

Systolic blood pressure is 105; *diastolic* 70.

Abdomen is distended and moderately tympanitic. There is no rigidity, no tenderness or signs of free fluid; no masses or viscera are felt.

Extremities.—No deformity, edema or tremor.

Lymph Nodes.—The posterior superficial cervical on the right side are slightly enlarged.

Pelvic examination not made.

Rectal examination not made.

Spine.—There is marked posterior spinal curvature of the "round back" type, but no angular deformity; no direct or indirect spinal tenderness.

Nasal Examination, 3/15/19 (by Dr. Coakley). A moderate amount of mucopurulent secretion in both nares, more in the right. There is a large post-operative opening in the nasal wall of the right maxillary sinus beneath the inferior turbinate. A trocar is passed into the antrum and as a probe feels thickened mucosa. On irrigating the cavity a small amount of the mucoid secretion came away. The swelling and redness over the upper part of the right maxillary region are not connected with the inflammation within the sinus.

Examination of the Eyes, 3/18/19 (by Dr. Shoenberg).

Visual acuity, left 20/20; right 20/40.

Motility of eyes is normal.

Both cornea anæsthetic, right complete, left incomplete.

Looking at the light with a red glass in front of the right eye she calls it brown; with a red glass in front of the left eye she calls it red. Looking at a piece of red

paper she says with the right eye "dark red," and with the left eye "light red."

A slight diplopia (homonymous) on looking to the right probably due to a slight deficiency of the right external rectus.

There is slight insufficiency of convergence.

Fundi normal.

Fields of vision markedly contracted.

CLINICAL NOTES.—3/18/19. This evening the patient became markedly excited and tried to get out of bed; restriction of no avail. She complained of pain in the suprapubic region; examination revealed a distended bladder. She was catheterized and a considerable amount of urine was obtained, but the pain was not relieved. She was given $\frac{1}{4}$ grain morphine by hypodermic, 10 c.c. paraldehyde, intravenously, and 1 grain of codeine, with only a little effect. Continued to be delirious during the night. No change in the reflexes; no other abnormal signs noted except an automatic movement of the right arm.

3/21/19. The patient is quiet and quite rational during the day time, but at about eight o'clock each evening she becomes noisy and irrational, complaining of headache, pain over the right cheek and difficulty in breathing. It requires 1/150 grain of hyoscine to keep her quiet.

LABORATORY EXAMINATIONS.—3/11/19. *Culture* from the right anterior naris gives a gram positive coccus, probably staphylococcus.

3/12/19. *Blood culture* is sterile.

3/12/19. *Wassermann*, both alcoholic and cholesterin antigens, negative.

3/12/19. *Blood Count*:

White blood cells	18,000
Polymorphonuclear leucocytes	85%
Large lymphocytes	3%
Small lymphocytes	10%
Eosinophiles	2%

3/15/19. *Blood Count*:

Red blood cells	4,740,000
Hemoglobin	85%
White blood cells	10,400

Polymorphonuclear leucocytes 65%
 Lymphocytes 33%
 Eosinophiles 2%
 3/15/19. *X-ray of skull* showed a slight amount of cloudiness in the lower portion of the right antrum which may in part be accounted for by the overlying petrous portion of the temporal bone.

3/15/19. *X-ray of spine* showed no evidence of tuberculosis.

3/16/19. *Sputum Examination*.—No acid-fast bacilli found.

3/17/19. *Spinal Puncture*.—Spinal fluid, amount 20 c.c. Appearance clear, colorless, pressure slightly increased. Lymphocytes, 2. Globulin absent. Reduction, Fehling's solution, present. Wassermann reaction; both alcoholic and cholesterin antigens, negative.

3/17/19. *Urinalysis*:

Specific gravity	1.015
Reaction	Acid
Color	Straw
Precipitate	None
Albumin	None
Sugar	None
Microscopical examination	Occasional white blood cells

3/18/19. *Von Pirquet test* was positive.

3/18/19. *Blood Count*:

White blood cells	10,500
Polymorphonuclear leucocytes	69%
Lymphocytes	31%
Eosinophiles	0%

3/19/19. *Sputum examination* for tubercle bacilli, none found.

3/21/19. *X-ray examination of the lungs* showed a moderate increase in density in the shadow of both lungs especially at the left apex.

4/1/19. *Blood Count*:

White blood cells	10,000
Polymorphonuclear leucocytes	71%
Large lymphocytes	3%
Small lymphocytes	15%
Large mononuclears	2%

Transitionals	3%
Eosinophiles	6%

Caloric Intake:

4/15/19	349 calories
4/16/19	1200
4/17/19	550
4/18/19	728
4/19/19	308
4/20/19	808
4/21/19	353
4/22/19	699
4/23/19	681
4/24/19	1304
4/25/19	2068
4/26/19	329
4/27/19	224
4/28/19	625
4/29/19	905
4/30/19	369
5/1/19	523
5/2/19	696
5/3/19	884

Average daily 716 calories

Weight on Admission,	3/11/19	110
	4/4/19	110
	4/17/19	109½
	4/21/19	110½
	4/27/19	112
Weight on Discharge,	5/5/19	113½

NEUROLOGICAL EXAMINATION, MARCH 15, 1919

VOLUNTARY MOTOR SYSTEM.—Patient is sitting propped up in bed with no evident signs of pain or discomfort. Breathing rapidly but with no apparent difficulty. The spine is uniformly bowed backward but without lateral curvature or any angular deformity. The abdomen is prominent and appears distended.

Gait.—Though the patient walked into the hospital, she now says she is afraid she cannot walk and on getting her out of bed she has great difficulty in standing without assistance. On attempting to walk she moves the left leg forward in a very unsteady manner and drags the right one

after it. She will not attempt to walk backwards or sideways.

Coordination.—On attempting to stand with the eyes either closed or open she sways in all directions and would fall if not supported. There is no non-equilibratory incoordination. On lying in bed the fingers-to-nose test, pointing and pass pointing, heel-to-knee, and "toe-to-object-above-recumbent-patient" test are well performed on each side. There is no dysmetria or adiadochokinesis.

Skilled Acts.—There is no aphasia or apraxia. The patient is able to salute, feed herself or arrange her hair. She has no difficulty in writing her name.

Reflexes:

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Jaw	Present	Present
Pectoral	Present	Present
Biceps	Active	Active
Triceps	Active	Active
Radial	Active	Active
Ulnar	Active	Active
Wrist	Active	Active
Suprapatellar	Active	Active
Patellar	Active	Active
Achilles	Active	Active

Reflexes:

<i>Superficial</i>	<i>Right</i>	<i>Left</i>
Ciliospinal	Present	Present
Supra-umbilical	Active	Active
Suprapubic	Active	Active
Upper lateral abdominal	Active	Active
Lower lateral abdominal	Active	Active

Plantar and its modifications

Plantar flexion	Absent	Absent
Babinski	Absent	Absent
Chaddock	Absent	Absent
Oppenheim	Absent	Absent
Gordon	Absent	Absent
Schafer	Absent	Absent

Abnormal Involuntary Movements.—There are no tremors, twitchings or choreiform movements.

Muscle Strength.—Patient is able to rise from the recumbent position to the sitting

posture. Opposed and unopposed movements of the neck, shoulder, arm, forearm, hand, finger, thigh, leg, foot and toes are all normal and equal on the two sides.

Muscle Status.—The volume, contour, consistency and tone of all the muscles seem to be normal.

Abnormal Associated Movements.—No abnormal associated movements are present.

NERVE STATUS.—Nothing noted.

GENERAL SENSORY.—There is an entire absence of the sense of touch, pain, temperature and posture on the right half of the body except for an area over the right malar bone where there is distinct hyperesthesia to both touch and pressure. The line separating the anesthesia from the normal part corresponds accurately to the exact mid-line. She is usually conscious of deep pressure over the muscles of the extremities, but at other times says that it gives rise to no sensations. She is unable to give the size, shape or form of any article placed in the right hand but is aware of the presence of some object in her hand and states that it feels "wooden." The sense of touch is absent from the right cornea, the right nostril, the right external auditory meatus, the right half of the buccal mucous membrane, the teeth and gums of the right half of the mouth, the right half of the tongue and all of the pharynx. There is also anesthesia of the right half of the vulva. All forms of sensation on the left half of the body are entirely normal except for the left cornea which is partially anesthetic, and for the left half of the pharynx which is mentioned above.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—There is an absence of the sense of smell on the right side, though it is normal on the left.

Optic and Oculomotor Apparatus.—Visual acuity is 20/40 in the right eye and 20/20 in the left. Both visual fields are

concentrically contracted. Both fundi are normal. There is no evident limitation of ocular movement though there is a slight diplopia in the right eye in looking far out to the right. There is also monocular diplopia when a small object is gradually moved away from the right eye to a distance of about fifteen inches. The pupils are regular, but unequal, the right being 2 mm. in diameter and the left 3 mm. The reactions to light and on accommodation are normal. The palpebral fissures are equal, each measuring 10 mm. The position of the eyeballs in the orbits appears normal. There is no nystagmus.

Trigeminal and Mouth.—The sensations of touch, pain and temperature are absent over the right side except over the right malar bone where there is hyperesthesia. Sensation is absent from the right buccal mucous membrane and right half of the mouth. All movements of the jaw are equal and normal on the two sides.

Facial and Face.—There is no facial weakness or palsy. Taste is absent from the anterior two-thirds of the tongue on the right side.

Acoustic and Ear.—At the time of this examination she seems quite deaf, but at some times she understands conversations in tones of ordinary intensity very easily. At present a watch is not heard at all in the left ear and heard but eight inches from the right ear. With a tuning fork air conduction is better than bone conduction on the right side. On the left side bone conduction is diminished and air conduction not perceived. Both ear drums appear normal to otoscopic examination.

Glossopharyngeal and Vagus, Pharynx and Larynx.—Salivary secretion seems normal. There is no deviation of the uvula to either side. Palatal reflexes are absent on each side; pharyngeal reflex is also absent. There is no aphonia and the quality of the voice is normal. There is no difficulty in swallowing.

Spinal Accessory.—There is no weakness of either trapezius or sternomastoid muscle.

Hypoglossus.—The tongue is protruded in the midline and its movements in all directions are normal. Articulation is normal.

During her stay in the hospital the patient has not seemed to improve in any particular, and in some ways became decidedly worse. On admission she walked into the hospital, but after being confined to bed for ten days, she was unable to stand or walk without support, though while lying in bed there was no muscular paresis and no incoordination of movement. Later a weakness of the right arm and leg appeared, which is evident in the photographs. Twelve days after admission retention of urine developed and subsequently constant catheterization was necessary.

There were rather frequent attacks of asthma which were usually of very short duration, sometimes of only a few hours. During the attacks there was hyperresonance over both lungs, sibilant and sonorous râles everywhere and prolonged labored expiration. These findings disappeared between attacks and there were at no time any signs indicative of tuberculosis. Adrenalin administered subcutaneously had no effect on the asthmatic attacks but they seemed to be relieved by 40 minims of paraldehyde in a teaspoonful of rhubarb and soda mixture.

DISCUSSION

There has always been a good deal of discussion as to whether there could be a fever of hysterical origin; cases of hysteria in which fever was present have been subjected to the criticism that some latent complicating infection was probably the cause of the temperature; and as these patients are not subject to autopsy it is an objection that has been very difficult to refute. It is well known that psychic disturbances may give rise to transient rises in temperature, but that a permanent pyrexia would be produced has been difficult to believe. The fact that fraudulent temperatures are at times given by hys-

terical patients, produced by friction of the thermometer bulb under the tongue, drinking of hot water before the temperature is taken, and again by applying the thermometer directly to a hot water bag, has also seemed to discredit the possibility of a true hysterical fever. Also the cases of hyperpyrexia where the temperatures reported were higher than is compatible with the continuance of physiological functions, have added distrust of the entire question. However, that there may be an abnormal elevation of temperature in this condition is recognized, and cases have been reported by Strümpell,⁶ Oppenheim,⁴ Kaufmann,² Osler,⁵ Larre³, Briand,¹ Voss,⁷ and many others. Manahiloff gave the subject a thorough critical analysis in a Montpellier Thesis of 1903, and concluded that there was unquestionably pyrexia of hysterical origin.

Hysterical fever as described by these authors has no constant characteristics, and its variations are as protean as are other manifestations of this disease. It may be continued, remittent or intermittent; it may be irregular, periodic or slow, but the intermittent or remittent form is probably the most common.

One of the most important distinguishing features of hysterical fever as against that of infectious origin is the absence of the usual parallel association of the temperature, pulse and respiration curves. This dissociation, however, is not always present.

Theoretically, fever may be due either to an increased heat production with normal heat dissipation, to normal heat production and diminished loss, or to a combination of increased heat production and diminished loss. It has been definitely established by calorimetry that the majority of all fevers—as those produced by specific infectious diseases and intoxications—are due to an increase in heat production, which is of course due to increased body metabolism as well as altered heat loss. The discrepancy between the assim-

ilation and the increased tissue destruction gives rise to the bodily wasting which is almost universally associated with continued febrile diseases.

This condition is a sharp contrast to what is found in the pyrexia of hysteria. Here the patient may have a temperature not unlike that seen in typhoid fever where the average increase in metabolism is 40 to 50 per cent, but without any perceptible tissue destruction or loss in weight even though the fever is present for very long periods. With an average normal caloric intake a continued fever which does not give rise to loss in weight cannot be due to increased metabolism.

In the second case reported above there has been constantly present a fever which ranges between 99° and 104° for a period of a year and a half, and during this entire time there has been no loss in body weight, while the calculated caloric intake during her stay in the hospital has not been greater than the average normal, being less than 1000 calories a day.

It therefore seems quite clear that the increase in temperature which is associated with hysteria cannot be due to an increase in metabolism.

Another striking difference between the hyperthermia in hysteria and a pyrexia of infectious origin is the entire absence of the phenomena which are so inseparably associated with our conception of fever that they are included in some definitions of it. The hysteric is as unconscious of her temperature of 104 or 105° as she is of her anesthetics. She does not complain of malaise, weakness, anorexia, thirst or vague pains in the back and limbs. The skin is neither abnormally moist nor dry and parched. The rapid elevation of temperature is not associated with a chill and there is no profuse perspiration when it falls to normal within a period of a few hours.

It therefore seems fair to assume that a continued elevation of temperature in a patient showing signs of major hysteria,

without loss of body weight, parallel quickening of the pulse and respiration, general weakness, or malaise, increased thirst and loss of appetite, or vague generalized pains, may be safely considered to be of functional origin and not due to some obscure infectious process.

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HYSTERICAL DYSBASIA *

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Hysterical phenomena have always been of such great interest to clinicians that an extended report of an unusual case is worthy of the closest study. The bizarre manifestations and the simulation of organic affections of the brain and cord have engrossed the minds of many clinical observers. Nor has the last word been said on the subject; for until we gain a clearer insight into psychogenic conduct, we shall not understand the peculiar vagaries of the hysterical make-up. The treatment of these conditions is not without its difficulties and rewards, as an abundant literature will readily testify.

The subject of this report is a boy 13 years of age, who comes under observation for a disturbance in gait and marked lateral curvature which has been in process of development since March, 1918.

Four months prior to the onset of this disturbance in his gait he had severe pains along the heel and external border of the left foot. This pain continued with varying intensity for four months, and in order to shield the affected part as much as possible he limped considerably. The limping continued and gradually became merged into the fantastic gait which he at present shows. The distortion of the spine evidently went on apace with the disturbance in gait. The patient thinks the condition is getting worse. His previous history furnishes us

very meagre information with regard to the development of his present condition. Two severe falls, the last one three years ago in which he was rendered unconscious, might be mentioned as suggestive data. He lives under a very good environment, and his home life is harmonious and free from any friction or restraint. He is an exceptionally intelligent boy who appreciates the abnormality in his gait and spinal deformity and evinces a keen desire to overcome both.

Family history is unimportant.

EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Abnormal attitudes and deformities.*—The patient shows an obvious spinal deflection which he can easily overcome or correct by voluntary effort, such as extending his hands above his head.

Gait.—Markedly incoordinated and has elements of ataxia, steppage and spasticity; is probably better appreciated when demonstrated than described.

Coordination.—All types of coordinative acts performed with perfect precision.

Dysmetria.—None noted.

Skilled acts.—Varied skilled acts performed in a normal manner.

Abnormal involuntary movements.—None noted.

Muscle strength.—Unimpaired for all types of muscular effort.

Muscle status.—No atrophy or hypertrophy, no hypotonia or hypertonicity.

Electrical reactions.—Normal for all types of modalities.

Reflexes.—Slight hyperreflexia of deep reflexes of upper and lower extremities.

Superficial reflexes.—Normal.

Pathological reflexes.—Absent.

Clonus reflexes.—Absent.

GENERAL SENSORY. *Acuity, localization and discrimination.*—Normal for all types of sensibility.

Muscle, joint and tendon sense.—Normal.

Vibratory sense.—Normal.

CRANIAL NERVES. *Olfactory and nose.*—Normal.

Optic and oculomotor apparatus.—Normal.

Trigeminus and mouth.—Normal.

Facial and face.—Normal.

Acoustic and ear.—Normal.

Glossopharyngeus and vagus.—Normal.

Pharynx and larynx.—Normal.

Spinal Accessory.—Normal.

Hypoglossus and tongue.—Normal.

MENTAL STATUS.—Excellent.

SYSTEMIC EXAMINATION.—Reveals no noteworthy abnormalities.

SUMMARY AND DIAGNOSIS

A critical analysis of the development of the bizarre gait and its associated spinal

distortion points unfailingly to a psychogenic type of disorder. The persistent neurologic data readily remove it from the organic affections of the cord and peripheral nerves. One is tempted to venture another diagnosis the clinical features of which simulate, in some respects at least, the case under discussion; that is, the torsion neurosis of Ziehen, or dystonia musculorum deformans, the etiology and pathology of which are still the subject of sharp division of opinion, some authorities contending that it is psychogenic while others are equally convinced that it has an organic foundation. It is merely mentioned in this connection as offering a fruitful field for discussion.

DISCUSSION OF TREATMENT BY DR. FRAUENTHAL

Dr. Frauenthal contends that the best way to obtain a cure in this boy is to make him walk up and down in front of mirrors so that he may view his grotesque behavior. The mental concentration, which is an essential part of the treatment, is thus readily obtained. Mild suggestion and repeated reference to the fallacy of his conduct as he views it himself in the mirror are brought into play, and eventually his resistance is overcome and a cure is effected. Mental concentration in these mirror exercises thus achieves a double object. The body as well as the mind is brought under its influence.

SOME COMMON DIFFICULTIES IN THE DIFFERENTIAL DIAGNOSIS OF HYSTERIA *

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In presenting the following case it must be mentioned that the disappearance of the patient before all the usual examinations could be obtained explains the lack of obviously necessary data; but while a definite diagnosis may be impossible from the material in hand, the findings afford opportunity for discussion, especially from psychogenic and therapeutic aspects.

Patient, E. C. (Case No. 23057); age thirty-five, female, married, white, born in the United States; housewife.

HISTORY. *Present Illness.*—The patient complains of "fainting spells" which began about three years ago. They have increased in frequency and now exhibit a convulsive form. On November 8, 1918, she applied to the medical department of the Vanderbilt Clinic, and was referred as a neurological case with the diagnosis of epileptiform convulsions. She dates the onset of her condition from March, 1916. She was sitting at the table eating when she became unconscious; her condition she states was like a "weak spell." She regained consciousness in about five minutes and except for weakness of short duration experienced no ill effect. There were no convulsive movements. The attack was not initiated by fright or any disturbing influence at the time so far as she knows. She volunteered the explanation, however, that her mother had recently told her, upon learning that she was pregnant, that if she should have another child "she ought to die having it," because her last two children had been sickly and had died. Subsequent attacks occurred about two or three times daily throughout pregnancy. In one week she had 29. Because of these attacks, for five weeks previous to her delivery she was

in a maternity hospital. Following the birth of her child the attacks did not recur for three months.

The patient describes the attacks as follows: "They start like something hitting me in the heart. It sounds like a hammer and I can hear it and it beats very fast. It seems just like an engine to me." The noise seems so real to her that she asked her family if they also hear it. The aura lasts from three to four minutes and the sensation is like a cold chill up the spine and to the top of the head where it is like fire through the head and the "top of the head seems to be moving up and down." Formerly when she felt the initial sensations she could get a drink of water and thereby as she thought prevent the unconsciousness. On these occasions she would be very weak and sometimes only feel cold. At other times she would be chilly and shake. She has been able to recall happenings in attacks but could not speak.

Upon two occasions the patient has had attacks in the clinic, one when she presented herself for the blood Wassermann test, and a second time while waiting her turn for an interview. The writer did not observe these convulsions, but from those who saw them the following data were gathered:

The body was rigid, evidently in tonic spasm, as efforts to extend the fingers were unavailing. The eyes were closed, but upon lifting the lids they were seen to be rolled back. The face was pale. There was no incontinence. The patient suffered no injury. About two minutes after the onset of one of the attacks electrical stimulation was applied and recovery was immediate.

Upon her next and last visit to the clinic Dr. McKendree succeeded in producing a

* Presentation of case report at the clinical conference of the Neurological Department of the Vanderbilt Clinic, November 25, 1918.

hypnotic state. With the patient recumbent, with eyes closed, in a darkened room, a vibrating tuning fork was applied to the lower limbs and she was told the spell would come on. Immediately she announced that it was coming and that she felt the beating at her heart. Panting respirations ensued with accompanying acceleration of the heart action. The condition lasted about a minute and was evidently dispelled by suggestion, for the patient opened her eyes directly when told to do so. The experiment was repeated immediately with similar but less marked reaction. The patient said the hypnotic state resembled the convulsions except that the beating was not audible to her and the sensations in the spine and head were absent. Following the recovery she felt the customary weakness and it was noted that her hand and arm trembled. She described a sensation of weight over her heart which evidently was due to the pressure of the writer's hand to determine the heart rate, but the patient did not identify it as a direct pressure from without.

For the past eight months she has experienced a dream state somewhat similar to a condition she had many years ago. Since she has been under observation for tuberculosis she has slept in an alcove and dates the disturbing dream from this change. She describes a form standing in her room at night and calling to her, "Come." She says she hears the voice and has asked members of her family if they also can hear it. The form seems to be herself and it recurs nightly in the same setting.

Previous History of Psychopathic Condition.—The patient states that her mother said of her that as a child she was always nervous and "whined." She was somewhat seclusive.

From seven to nine she lived in the home of an aunt, and her nervous condition was so aggravated by the hysterical outbreaks of this relative that her mother took her home. The patient said that when such

outbreaks occurred, she would jump up and down and scream with fright.

Over a period of four years ending at twenty years of age when she was married, the patient was subject to attacks of somnambulism which occurred every two or three months. These experiences the patient recalled only as a dream upon awakening the following morning. The dream content initiating the somnambulistic state was always the same. She saw her father (who died when the patient was five years old) standing by the foot of her bed. She would scream aloud and get out of bed, whereupon the family was aroused and her mother would take her to bed with her. The dream continued until she slept again. The father's form followed her as she went to her mother's room and there stood by the bed. The patient would ask her mother if she also did not see it. She also complained of feeling cold, but was amnesic for the conversation and the sensation of chilliness, though to relieve the latter the mother always supplied extra cover. She did recall her mother's urging her to go to sleep.

There was no recurrence of somnambulism from her marriage until eight years later. From then until three years ago these states were frequent but they were unaccompanied by dreams.

Previous History.—There are no data of birth, infancy and development. The patient reached the fifth year in school. Her habits show no excesses. During her third pregnancy her weight reached 200 lbs. Now it is 134. Her weight with the exception of the above has not exceeded 140 lbs. Apart from frequent vomiting throughout this pregnancy she had no abnormal complaint. Menstruation has always been irregular, varying in intervals from one to six months, but otherwise normal. For the past year she has had painful urination. Her libido is apparently much diminished. There is no history of injury or of any operative treatment. In childhood she had epistaxis and has always had dizziness. Syphilis is denied. She has

had a vaginal discharge for the past year. For the past six months she has been reporting to a Board of Health clinic for tuberculosis. Recently a tooth has been the seat of an abscess.

Family History.—The patient has had six children. She had a threatened abortion in the third month of the last pregnancy. Her first child died of meningitis at two years. The second is 12 years of age and well. Her third pregnancy was marked by vomiting throughout and a marked excess of weight (200 pounds). This child has a marked defect in speech and at nine years of age is in the second grade in school. The fourth child was a "blue baby" and died at one year. No definite cause of death was given other than that it did not develop. The fifth child died at nine months of "gastritis." The sixth pregnancy was marked by the convulsions of which the patient now complains, from the fourth month on. The child is now two years old and apparently normal. The patient's father died at 33 years of age of blood poisoning, her mother is living and in good health. A paternal aunt had hysterical attacks. There are two sisters and two brothers and one half sister, all living and well. The patient's husband is said to have a rash and patient's mother suspects he is diseased. The husband's brother has epilepsy.

VOLUNTARY MOTOR SYSTEM. Attitudes.—There are no abnormal attitudes.

Gait.—The gait is normal.

Coördination.—Coördination is not impaired.

Skilled Acts.—Speech is normal and there is no evidence of aphasia. Further data not obtained.

Abnormal Involuntary Movements.—A tremor rather coarse and irregular shows in the outstretched hands, which was more marked in the right side.

Reflexes.—(1 signifies slight, 2 active, 3 very active, 4 with clonus, 0 means absent.)

	Right	Left
Biceps	2	2
Triceps	2	2

Suprapatellar	4	3
Patellar	3	3
Achilles	2	2

Superficial reflexes.—

	Right	Left
Upper lateral abdominal	2	2
Lower lateral abdominal	not obtained	
Plantar flexion	2	2

Muscle Strength.—The muscle strength in the arms is apparently normal.

Muscle Status.—No record made.

Abnormal Associated Movements.—No record made.

NERVE STATUS.—No record made.

GENERAL SENSORY. Touch, Acuity, Localization and Discrimination are apparently normal on the body, face, and extremities.

Pain.—Superficial pain sensation is normal over the body, face, and extremities.

CRANIAL NERVES.—No recorded data except for the second cranial nerve which is as follows: Pupils are one-third dilated, equal, round, and centrally placed. The reaction to light and convergence are normal.

MENTAL STATUS.—The general intelligence is average. The attention is fair. The emotions are fairly well controlled. No memory defect was noted. No history of phobias or compulsions was elicited. In dream states the patient reacts to hallucinations of sight and hearing.

CRANIAL MORPHOLOGY.—No record made.

SYSTEMIC.—No abnormality was noted in the tegumentary or glandular systems.

Cardiovascular.—The pulse rate was 90 per minute. The area of cardiac dullness is within normal limits. There is a systolic murmur indicating mitral insufficiency but there are no evidences of deficient compensation.

Respiratory.—No râles are present but the normal vesicular breathing is replaced by harsh sounds over limited areas.

Gastro-intestinal.—Deep pressure elicits tenderness in the left lower abdominal quadrant.

Genito-urinary.—No examination made.

Skeletal.—No abnormality noted.

LABORATORY TESTS.—Wassermann of blood negative.

GENERAL SUMMARY

The prominent findings of the case may be grouped under the following headings: (1) heredity and environment; (2) physical condition; (3) somnambulistic states and convulsive seizures.

1. A paternal aunt was evidently a subject of hysteria. While residing with this aunt the patient suffered fright on numerous occasions. The patient also had a residence of about six months with an epileptic brother-in-law who suffered severe and frequent seizures.

2. While there is at present no evidence of tuberculosis, her history of attendance at a clinic for tuberculosis for the past six months bears evidence of probable pulmonary involvement.

Cardiac examination shows mitral insufficiency which, however, is fully compensated.

There is a probability of syphilis based upon the history of her husband's condition (rash) and the history of poor development and death within the first year of two children. The negative Wassermann and the history of a subsequent normal child tend to outrule syphilis.

3. The patient was a nervous child. From her sixteenth year to her marriage at twenty years of age she was subject to somnambulistic states. Somnambulism recurred eight years after her marriage and continued until three years ago, since which time she has been subject to convulsive seizures which are progressive in number and severity. The convulsions at the onset were characterized by partial loss of consciousness; now the patient believes she is completely unconscious. The patient never injures herself in attacks and is not incontinent. According to the patient's own statement the condition induced by suggestion was similar to the attacks she suffers. In this induced condition her

amnesia was incomplete, as evidenced by her remembrance of pressure exerted over her chest.

DIAGNOSIS.—If the conclusion of Babin-ski be true that hysteria can be diagnosed positively if the hypnotic state can be produced by suggestion and removed by persuasion, the experiments in hypnosis in this case made by Dr. McKendree establish the diagnosis. The patient's own statement of similarity of body sensations in the actual and induced states might be sufficient to exclude a coexisting epilepsy.

DISCUSSION OF DR. JAMISON'S CASE FOR DIAGNOSIS

MRS. DWIGHT, of the Social Service Department.—On November 23, 1918, at the request of the Neurological Department of the Vanderbilt Clinic, I visited this patient at her home, and found the family, consisting of husband, wife, and three children, occupying a five-room flat, two rooms of which are light, the remainder dim and opening on an air shaft. The rooms are very unsanitary, are infested with rats and always have a bad odor when closed for any length of time. The patient was at work in the kitchen and, although the weather was clear, all the children were at home, the mother being afraid to have them on the street as the children of the neighborhood are very rough. All three children are pale and very nervous. Willy, nine years of age, is a pale, thin boy, with a marked speech defect. He seldom plays with other children as they mimic him and laugh at him. He is a grade behind the average in school. Linda, 12 years of age, is also a grade behind her age in school. On two occasions she has fainted on her way home from school and each time was carried into a house to recover; on the following day she had a headache. The baby appears to be delicate, an attack of pneumonia last summer having nearly proved fatal. The husband, who is a wire worker, earns 20 dollars a week. From October,

1917, to June, 1918, he was unable to work following an infection of both feet from an operation for ingrowing nails. During this time the mother took in washing and worked night and day to support the family.

The patient gave the following history. She was one of six children. When very small she was sent to live with a widowed aunt who was subject to hysterical attacks, and the child was often alone with her at the time they occurred. She can remember calling for help when scarcely old enough to talk. Following these episodes, the child would "shake all over and cry." When nine years of age she was sent to a convent where she remained until the age of 17, when she came home and later went to work as a labeler. She married at 20 years of age and has had six children, three of whom are dead. The second child was born after she had been married eight years and lived only a year. This was a great shock and the mental picture of the child as he lay dead was always with her. Whenever she entered the room where he died she saw him and two months later, when attending a moving picture show, she saw him on the screen in the picture and cried out and had to be taken from the place. Three years later she moved from the house with her family to escape from the persistently haunting vision. She has had many falls about the home and in the street, an ambulance having taken her home a number of times. She sleeps in the front room of the flat, the bed facing an alcove, and at night she sees a vision of herself, all in white, holding out its arms and saying "Come to me." She cries out and awakens her husband who tells her she is crazy. She says she thinks this portends trouble.

DR. ADOLPH STERN.—I believe that the diagnosis of hysteria is correct in this case, irrespective of the previous history. We are justified in making a diagnosis of hysteria on the clinical findings, and not necessarily on the previous history or by exclu-

sion; the symptoms in this case justify the diagnosis of hysteria. That diagnosis was also proven correct by the patient having been put into a state of hypnosis, during which the symptoms were reproduced. Here we have a condition of hysteria in an individual who evidently, from her previous history, was a suggestible individual surrounded by an environment conducive to a further hysterical state. In addition she had seen her aunt during epileptic attacks, which may have had a great deal to do with the nature of her own hysterical manifestations. I think a very suggestive etiology for the onset of the condition is the warning of her mother that the patient might die if she bore any more children. Steckel has called attention to the predominance of criminal impulses in those individuals who develop epileptiform attacks in hysteria, and it may very well be that the warning of the mother that she might die if she gave birth to other children may have aroused a desire to get rid of this child, and during such moments the hysterical attacks came on. In this connection the sensation of anxiety located in the heart might emphasize this. When we are disturbed, especially by fear or anger, we often locate the feeling in the heart. That may be the origin of the hysterical cardiac sensations in this patient. I believe examination in this direction will elicit some unconscious impulses, the clearing up of which may help the patient.

DR. MORRIS GROSSMAN.—I saw this patient in an attack last week, while she was sitting awaiting her turn for therapy. I do not know how long the seizure lasted before I saw the patient, but it could not have been very long. She was lying on a bench, her eyes were closed and her teeth were tightly clenched, her arms and legs were somewhat stiffly held, and there were no clonic movements in any of the extremities. Examination of her pupils showed that they were normal in size and reacted promptly to light and her deep reflexes

were all present and equally active. Pressure over the supraorbital nerves elicited a grunt of pain with protest against this manoeuvre. As the case was lacking in all the cardinal elements of a true epileptiform convulsion I decided to try electrical stimulation as a test for the depth of unconsciousness, stating aloud that one or two minutes of electrical stimulation would bring her out of her seizure. With the first touch of a moderately strong galvanic current the patient promptly sat up and began to protest against further application of the electrical current. There was no confusion or drowsy state present, such as follows a true epileptiform seizure. The pupillary and deep reflexes did not show any change from the normal during or directly following the attack. There was no doubt in my mind that the patient was suffering from hysterio-epilepsy and not from true epileptiform seizures.

DR. LOUIS S. ARONSON.—I do not think that a single negative Wassermann in this case should exclude syphilis. The history is strongly suggestive of specific disease—a threatened abortion, three children dying in infancy, one child mentally and physically defective, and in addition the husband suffering from a disease that is suspicious. In my opinion a provocative blood, or better, a spinal Wassermann should be done in this case. As to giving the condition a name, the best name I can think of is

to go back to Charcot's hysterio-epilepsy if the case is not luetic.

DR. FREDERICK TILNEY.—In this connection I would like to make an observation with reference to the reflexes in the major epilepsies. Professor Lambert and I had occasion to study a case of compression of the brain as the result of an enormous abscess and we went into the matter of the reflexes very carefully. We found the patellar reflexes on both sides quite active while the patient was in her interval, but when she was about to have a convulsion this would cease and one minute and ten seconds afterwards the knee jerks would disappear, remaining absent for a few moments and then reappearing. She had a bilateral Babinski right along, but during the periods of convulsion it disappeared, coming back four or five minutes later. The Achilles disappeared later than the knee jerks, and the reflexes disappeared from the upper extremities so that it was absolutely impossible to bring them out. The pupillary reflexes were abolished at the end of a minute and returned within four or five minutes. This is an interesting point in differential diagnosis, the reflexes disappearing not immediately after the convulsion but after an appreciable lapse of time and remaining absent for four or five minutes. Their remaining present in this case of Dr. Jamison speaks against it being a true epilepsy.

THE CONTROLLABLE ETIOLOGICAL FACTORS IN INSANITY, AND HOW TO UTILIZE THEM

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In discussing the various etiological factors in the different types of abnormal behavior in which the personality of the individual is lost, it must be remembered that in the present state of our knowledge the causes appear so multiform and variable, so vague and subtle, as to be of doubtful value in determining the prophylactic measures in many of the psychoses. And yet there are sufficient available data that offer distinctly logical and valuable deductions utilizable in the prevention of insanity.

It is now universally acknowledged that except for the potentiality to acquire knowledge there is nothing in the mind but that which comes to it through the sense organs. The sensations are interpreted by the sensorium into perceptions. The affects, or the pleasant or unpleasant qualities which accompany any mental process, determine differences of value among the perceptive mass. Through thinking and ideation, the sensory and perceptive material is constellated, or arranged in a definite and useful way. The interaction of constellations produce conduct.¹ In the synthesis of constellations which results in the personality or mental make-up of the individual, environment, education, general health, and normal habit reactions play important and distinct rôles. The function of the mind is so to regulate and control the conduct of the individual as best to adapt him to his environment.² While the function of the mind is fairly well understood, there is yet to be found an adequate definition for mind itself. To the upper cellular layers of the cerebral cortex, the so called supra-cortex,³ are assigned the highest functions of the mind, such as the passing of judgment,

thinking, ideation, or the proper synthesis of constellations.

Bearing in mind that the physical and mental processes are interdependent and not distinct, still for practical purposes, the psychoses may be divided into the organic and the functional, the former resulting from anatomical alterations in the brain caused by chemical or bacterial toxins, and the latter resulting from abnormal psychogenic reactions to which there are no known pathological changes in the nervous system bearing a causal relationship.

THE ORGANIC PSYCHOSES

1. *General Paralysis*.—As a consequence of the successful invasion of the cerebral cortex by the spirochæta pallida, there results a progressive deterioration leading to a complete undermining of the whole mental and physical personality. This disease is responsible for about 14 per cent of the admissions to the New York State Hospitals for the Insane, and accounts for about 18 per cent of the deaths occurring in these institutions.⁴ The economic loss to the State of New York on account of the syphilitic mental diseases during 1917 has been estimated by the State Statistician at nearly five million four hundred thousand dollars.⁵ The amount of suffering caused by the disease is incalculable. We have then a condition in which the etiological agent is known, the pathological and clinical pictures thoroughly known to every medical man, and the therapeutic agents often proven efficacious. The State is entirely too parsimonious⁶ in applying scientific principles, and in view of this short-sighted policy the parietic is at present receiving, with a few glaring ex-

ceptions, only custodial care.⁷ This disease should be treated with salvarsan and mercury, as the parietic should not be deprived of a trial with those remedial agents that are at present recognized as specifics against syphilis. With proper prophylactic measures this disease could be made a medical rarity. Every case of primary or secondary syphilis should be regarded as a potential case of paresis. A thorough and systematic course of treatment which should cover a period of at least three years in the average case, followed by yearly examinations of the blood and spinal fluid, would materially reduce the number of cases of paresis. The application of prophylactics after exposure to infection has proven efficacious in preventing the occurrence of the disease among the soldiers and the sailors. Its efficacy is no longer a matter of speculation. We ought to instruct the public in the use of proper antiseptics and thereby lessen so much of human misery as is caused by the syphilitic virus. Of course we should not abandon the general sociological principles at our disposal; and the encouragement of early marriage, and emphasis on the holiness and purity of the family, are measures always within reach of those interested in the amelioration of ailment among human beings.

2. *The Alcoholic Psychoses.*—The effects of alcohol upon man are well known. Its relations to mood reactions have long been understood. It is a fact that the largest consumption of alcoholic beverages occurs whenever there is an alteration from the normal mood reaction, i.e., in times of joy and in times of sorrow. Very few religions have failed to incorporate the use of alcohol products in one or another ceremony. The recent congressional investigation in Washington has revealed some of the subtle ways by which dealers in alcoholic products have been taking advantage of the weakness of human nature and have been making capital out of it. While only about 6 per cent of admissions to the State Hospitals⁴ are placed under the cap-

tion of "alcoholic psychoses," alcohol is responsible for 25 per cent of all cases of insanity, as it is a potent factor in precipitating other psychoses. Alcohol is a depressant from the very beginning and has no stimulating action except in certain well-defined medical condition. Prohibition should offer the solution to the problem. The efficacy of prohibition might be gauged by the lessened number⁸ of alcoholic psychoses now found as a result of the increased price of distilled liquors. Anyone who teaches medical students realizes how difficult it has become to demonstrate a case of multiple alcoholic peripheral neuritis, etc.

3. *Psychosis with Cerebral Arteriosclerosis.*—In this condition the resulting abnormal behavior is due to vascular disease. While cerebral arteriosclerosis may occur without any coexisting general arteriosclerosis, in the vast majority of cases there is a coexisting general arteriosclerosis.⁹ The causes of arteriosclerosis are many and varied, and none as yet has been quite specific; endogenous and exogenous toxins, renal disease, gastro-intestinal auto-intoxication, prolonged mental and physical work, worry, and excessive ingestion of food, have been blamed for it. The presence of vascular nephritis found in many of these cases that come to autopsy, and the existence of edema around the blood vessels of the brain, must be responsible for not a few of the symptoms of this disease. There is no doubt that a blood pressure of over 150 mm. of mercury is a danger signal in the vast majority of people, unless there is a distinctly physiological explanation for the high blood pressure. The judicious use of the iodides, the treatment of existing renal insufficiency, the combating of intestinal putrefaction, and the leading of a life of moderation, will prevent many of the cases from becoming so serious as to cause psychotic symptoms.

4. *Senile Psychosis.*—It must be admitted that in this type of abnormal behavior the evidence of an existing etiological factor is not very convincing. How-

ever, the associating neurofibrillar alteration and the senile plaques found in the vast majority of brains from the senile psychotics¹⁰ must be considered as more than incidental.¹¹ There is usually found in these cases a concomitant cardiorenal insufficiency demonstrable in a chronic diffuse nephritis and a chronic parenchymatous myocarditis.⁹ The significance of neurofibrillar degenerations and miliary sclerosis is not as yet well understood. That they represent the results of the action of bacterial toxins upon nerve tissue, or that possibly they are the end results of the action of endogenous poisons, or that they are the expression of an abiotrophy of the nervous system, are views held by those who have studied this condition clinically and at postmortem examinations. To train the individual to approach the senium in a well prepared manner, is one of the functions of the modern hygienic movement. The investigation of the efficiency of the cardiorenal system should be resorted to at least once a year under the guidance of the family physician. Proper hydrotherapeutic measures, care of the skin and of the bowels, and attention to the cardiovascular and cardiorenal systems, are the logical available prophylactic measures to which we may resort in the present state of our knowledge.

5. *The Psychoses with Other Somatic Diseases.*—Under this heading are included those forms of abnormal behavior which are manifested during, or immediately following, the course of any somatic disease, such somatic disease being apparently responsible for the development of the psychosis. Classical examples are found in the infective and toxic exhaustive psychoses following streptococcus throat infections, influenza, pneumonia, puerperal sepsis, typhoid fever, pellagra, etc. Anatomically this condition is an expression resulting from a true injury to cell bodies of the cortical neurons. Typical cloudy swelling, chromatolysis, eccentrically situated nuclei, vacuole degeneration, and poorly staining protoplasm, are the

usual pathological changes seen in the cells of the cortex. The intensity of the pathological process in the cells of the cortex depends in a general way upon the intensity of the toxemia and upon the acuteness of the somatic disease. The clinical picture is usually that of delirium, hallucinations, and delusions, added to the ordinary picture of the particular somatic disease. To meet this type of psychosis it is important to treat the original somatic disease conscientiously. We are really in a difficult situation. It is apparent that some nervous systems do not stand the effects of fever as do others. The poorly nourished states of most of these patients would immediately suggest the necessity of forced feeding in the predisposing somatic diseases. Special attention to the bowels and great effort at checking the toxemia will help to prevent the occurrence of this type of psychosis.

THE FUNCTIONAL OR PSYCHOGENIC PSYCHOSES

Under this caption are considered those forms of abnormal behavior for which at present there are no adequate explanations found in the structural alterations in the brain. In the vast majority of cases coming to autopsy, the nervous system is found to be normal, or if any pathological conditions are found they are explained as the results of some concomitant pathological process. In the present state of knowledge concerning them, they are best explained as reaction types resulting from vicious habit formation¹² caused by bad mental hygiene in an individual of a distinctive mental make-up, characterized generally either by a reversion to an infantile form of behavior in the schizophrenic group, or by extremes of the normal form of mood reaction¹³ in the cyclothemic type.

1. *Dementia Præcox* (schizophrenia).—This type of abnormal behavior is responsible for 20 to 25 per cent of admissions, and for 55 to 60 per cent of the residents in the State Hospitals for the Insane.⁴

In these cases the behavior is of a distinctly infantile type. The individuals susceptible to it are those possessing the so-called shut-in personality.¹⁴ This personality is characterized by sensitiveness, stubbornness, reticence, seclusiveness, inability to adapt oneself to new situations, and inability to make friends readily or to participate in wholesome forms of diversions. They live in a world of dreams and phantasies rather than in a world of facts and actualities. They meet new conditions by shirking or even evading them, and are engaged in thought rather than in action. They do not discuss their difficulties with anyone and usually brood over them. As long as the situations demanding adjustments from them are of simple form they handle them fairly satisfactorily; but as soon as they are called upon to meet more complex conditions, they react in an infantile manner, and their behavior then assumes the characteristic psychotic aspect. As the demands for adjustments in the sexual sphere are the most complex that one is called upon to make, these peculiar individuals not infrequently break down in their effort to handle some one or another of their sexual problems. In the early manifestations of the disease we see opportunities for family physician⁸ and also school teachers to perform great service, as they are best situated to notice any peculiarities in the conduct of the child. If discovered sufficiently early, these children exhibiting them could be taught to react in a more wholesome manner and to meet problems and even difficulties frankly and openly. Their education could be so moulded as to prepare them for the work for which they are most fitted by their peculiar personalities, and thereby lessen qualitatively and quantitatively the adjustments they may be called upon to make later in life. This is especially true of the high school and college period of life, when many of these individuals are beginning to manifest peculiarities of conduct apparent to the average teacher and even to the layman. They could be

taught such useful occupations as would bring them into the smallest number of difficulties possible and at the same time enable them to be self-supporting. Sex hygiene, if properly taught, would be of tremendous help to them in understanding the significance of many of their odd ideas and sensations, and would enable them to overcome many of their difficulties, especially if the latter were of the early and elementary types.

2. *Manic Depressive Psychosis (Cyclothemia)*.—This group comprises about 15 to 20 per cent of admissions to the state hospitals and about 30 per cent of admissions to the private institutions for the insane.⁴ This group, comprising essentially benign affective psychoses, occurs in individuals of a typical mental make-up.¹³ Individuals possessing such a make-up manifest it through manic or through depressive tendencies, by being overactive, vivacious, high-strung, very enthusiastic, inclined to overdo things, apt to undertake many duties and perform only a few, and sometimes very excitable; or on the other hand by showing a tendency to worry over insignificant matters and brood over trifles, by being subject to blue spells, slow in their physical activities and rarely happy. When these individuals suffer from failure of compensation for some unusual demand for adjustment and become psychotic, the symptoms which they manifest are really accentuations of the peculiar characteristics of their mental make-up. An element which has been rather undervalued as an etiological factor in their mental break is the one of fatigue. Whenever the katabolic process exceeds the anabolic process for a considerable period of time, fatigue occurs. In the case of the nervous system, fatigue may occur even when the rest of the tissues and organs of the body are in a condition of apparent rest. In many of the carefully elicited histories of these cases, there is considerable evidence of the great stress and tension to which the nervous systems have been subjected for a long period of time and

immediately before the onset of the psychosis. The stress usually has been in the shape of prolonged physical, or more often prolonged mental, work, worry, anxiety, or dissipation. These individuals if taken in hand early by properly trained physicians and teachers could be taught to moderate their emotional reactions and to check any manifestations of abnormal mood reaction and behavior. They should also be taught the proper way to lead a life of moderation, and the means of adjustment to the vicissitudes of life. They should not be allowed excesses of any sort. The individual with a maniac make-up should be taught the value of concentration, and the one with a depressed make-up should be initiated into the pleasures that come from a diversity of interests.

THE PROBLEMS OF THE DISCHARGED PATIENTS

These represent individuals who, having had a decompensation of their psychological efficiency, have shown sufficient psychotic symptoms to warrant society in segregating them, and because of treatment or lack of treatment, or sometimes in spite of treatment,¹⁵ have regained sufficient mental compensation to be allowed to return to their former circle of society. It is in these cases especially that we must not lose sight of the fact that one may be mentally ill and still be considered well in the eyes of the law, that one may be psychotic and at the same time sane, and that insanity is a legal rather than a medical term.³ We have the discharged patient and the same environmental conditions that existed prior to the onset of the psychosis, and the handicap that comes from loss of confidence by the patient, as well as the prejudice of society against the recovered psychotic individual. To meet these conditions we must have the cooperation of the social service worker and the physician, and also the assistance of popular education. The social service worker could so arrange the affairs of the indi-

vidual as to elicit from him the least demands for adjustment. It is a fairly good principle to change the environment of the individual whenever possible, and allow him to live at least in new surroundings thereby minimizing the possibility of recurrence of such environmental conditions as may have shared in precipitating the original mental break. The benefit derived from change of surroundings, even if it be merely in the change of wards, is one universally acknowledged by those who have had experience in the treatment of psychoses.¹⁶ The physician must follow up the case in order to insure against a return of the symptoms. It is preferred that the same physician who has been treating the patient at the hospital should follow up the case, for the confidence which the mental patient has learned to place in his physician is far above the confidence which an ordinary patient places in his physician. In order to follow up the cases it is important to have dispensaries where the patient may come and there meet his physician. These dispensaries could serve the additional valuable¹⁷ purpose of serving as a place of refuge for those in the prepsychotic period of their mental ailment or the stage of threatened failure of mental compensation. The education of people at large to a realization of their duty in checking the spread of mental diseases has gained impetus during the war through the publicity given by the lay press to "shell shock" cases, and much benefit may be expected to come from this.

SUMMARY

1. In the formation of the personality of any individual, environment, education, the general health, and normal habit reaction play distinct and important rôles.
2. The solution of the problems of syphilis and alcohol would reduce by at least one-half the number of cases of insanity. The application of antiseptics after exposure to infection, through treatment

of the early stages of syphilis, and prohibition of the manufacture of distilled liquors, are the logical method of attacking these problems.

3. A systematic yearly physical examination by a physician, with special attention to the conditions of the cardiovascular and of the renal systems, would diminish the frequency of the arteriosclerotic and senile psychoses. The thorough treatment of infectious diseases would prevent the occurrence of many infective exhaustive types of psychosis.

4. Checking abnormal mood reactions, and preventing the formation of vicious habit reactions, would minimize the occurrence of the psychogenic psychosis.

5. The establishment of properly equipped dispensaries for treatment of the mentally ill, the organization of psychiatric social service departments, and popular education in the elementary mental hygienic principles, are essential in preventing the spread of mental disease.

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ON THE BABINSKI PHENOMENON IN ANTERIOR POLIOMYELITIS *

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The subjoined case is reported because it showed the Babinski toe phenomenon not commonly seen in anterior poliomyelitis. The case is further of interest in that there was probably a Babinski on the non-paralyzed side.

There are many neurologists who look with suspicion on the reported presence of a Babinski in a pure poliomyelitis, at times even questioning the accuracy of the observation. Nevertheless there are undoubted cases of poliomyelitis with dorsal extension of the big toe resulting from trying to elicit the Babinski phenomenon or its modifications. Oppenheim is of the opinion that the dorsal extension is not a true Babinski, and he explains it on the ground of preserved strength or lesser paralysis of the extensor hallucis longus and complete paralysis of the other flexors and extensors. That is, this single muscle alone being preserved, it will respond to any skin stimulation, and that response being an extension of the big toe it simulates the Babinski. It is quite possible that this explanation holds in some cases, but it is not true in all. The electrical reactions should decide this point in any doubtful cases.

But there are other possibilities which perhaps more nearly explain the probable pathological origin of the Babinski in poliomyelitis. While we do not exactly understand the Babinski, we do know that it is a sign of pyramidal tract involvement anywhere in the course of the primary motor neuron from cortex to secondary motor cell. It has also been fairly well proved that the lesion in poliomyelitis is far more extensive than the clinical signs and symptoms usually indicate. While

the inflammation does severely affect the anterior gray horn, it also extends to the columns of white matter. Indeed there are cases which have shown almost complete involvement of the whole cord with comparatively few paralytic phenomena. One need not, therefore, do great violence either to credulity or imagination to presume an injury to the pyramidal tract as an explanation of the Babinski. That it does not often occur is no reason why the inflammation should not occasionally extend to the lateral column and compromise the pyramidal tract.

But there is still another explanation. We know that poliomyelitis is frequently complicated by brain involvement; and indeed frank cases are diagnosed as polioencephalitis. But there are cases with probable encephalitic foci which are not frank; that is they do not show the usual cranial nerve paralyses which serve to distinguish and localize the process. However, a focus in a so-called silent area or somewhere along the course of the cortico-spinal tract may complicate a poliomyelitis and perhaps not attract attention. It is in such cases that an unheralded focus along the pyramidal tract leads to the subsequent detection of a Babinski, and it becomes striking when it occurs on the paralyzed foot.

There is a fourth possibility. The poliomyelitic focus in the anterior horn may extend to the anterior column and involve the direct cortico-spinal fibers and thus give a Babinski. Obviously the toe phenomenon will be not on the paralyzed but on the opposite side. In the *Wiener klinische Wochenschrift* for 1905 Dr. Schiller reported three cases of poliomyelitis with

* Presented at the Clinical Conference, March 6, 1919.

Babinski on the paralytic and non-paralytic sides, and he mentions the fact that Rothmann produced experimentally a lesion in the lumbar segment of the cord in dogs, which included the anterior horn, anterior and lateral columns. The result was paralysis of the limb, Babinski on the same and opposite sides. It is quite possible, then, for an actual focus to correspond to an experimentally induced lesion and thus give rise to a Babinski.

CASE NO. 23951. K. S., school boy, aged sixteen, Syrian by birth, gave as his main complaint pain in the middle of the back. He attributed this to a fall on his back which he sustained in 1915. He was not unconscious or even seriously hurt, but since then pain gradually set in, which has become steadily worse. It is a dull pain, not very severe at any time nor ever sufficient to incapacitate him. It does not radiate. He also stated that a curvature of the spine began to develop following the fall. There were no other complaints. His habits are good, he eats and sleeps well, his bowels are regular, there are no urinary disturbances; no cough or dyspnea, no visual disturbances either past or present, and no headache. His early history is negative except for the fact that when one year old he had an infantile paralysis which involved the right upper extremity and left lower. Both limbs have remained severely affected to this day. Of operations he had a tonsillectomy and circumcision. The family history is negative, the siblings being normal and healthy.

VOLUNTARY MOTOR SYSTEM. Abnormal attitudes and deformities.—Kypho-scoliosis to the right; pes cavus left foot, flat foot right and left drop foot. Right arm hangs by the side.

Gait.—Steppage left; hip phase left.

Coordination.—Normal equilibratory; non-equilibratory normal on the left upper right and right lower extremities. Tests could not be made with right arm and left

leg owing to the paralysis. Skilled acts impossible of execution with right hand. Speech normal. No abnormal involuntary movements.

Reflexes.

<i>Deep</i>	R	L
Pectoral	I—	I
Biceps	?	I
Triceps	?	I
Radial	?	I
Ulnar	?	I
Suprapatellar	0	0
Patellar	I	0
Achilles	2	?
Periosteal	0	0

<i>Superficial</i>	R	L
Ciliospinal	I	I
Supra-umbilical	2	2
Suprapubic	2	2
Upper lat. abdom.	2	2
Lower lat. abdom.	2	2
Cremasteric	I	I
Plantar flexion	?	0
Babinski	P(?)	P
Chaddock	0	P
Oppenheim	0	0

Muscle Strength.—The patient has to support himself with both hands in sitting up from recumbent position and climbs up on himself in standing up. The infantile, quadrupedal way of getting up reminds one of muscular dystrophy. Of individual muscle there is marked weakness in the right shoulder, arm, forearm and hand, weakness of the flexors of the left leg and plantar flexors of the left foot. There is almost complete paralysis of the left quadriceps and dorso-flexors of the foot. There is atrophy both in volume and contour of right upper and left lower, the right arm being five centimeters less in circumference than the left, the left thigh three centimeters and left leg half a centimeter less than their corresponding limbs on the other side. Marked hypotonia was indicated, and normal irritability but no myo-edema of affected muscles. Owing to failure on the part of the patient to keep several appointments no electrical

reactions were taken. There were no abnormal associated movements.

NERVE STATUS was normal.

GENERAL SENSORY.—Touch, pain, temperature, vibratory, muscle-tendon and stereognosis normal all over.

CRANIAL NERVES.—Olfactory normal. Optic, oculomotor, trochlear and abducens. Vision, fields and fundi normal. Pupils normal in shape, size, position and reaction to light and convergence. There was questionable strabismus, the right eye being slightly turned out, and it did not converge quite properly. The patient thought he had had this condition since infancy. There was doubtful nystagmus on looking to the right. Palpebral fissures normal. All the other cranials were normal. No diminished corneal or pharyngeal sensation.

MENTAL STATUS was normal.

SYSTEMIC.—Abundance of hair all over the body. Slightly enlarged thyroid. Heart and lungs normal. X-ray of spine shows a plain kypho-scoliosis. Blood Wassermann was negative.

DIAGNOSIS.—There seems to be no doubt that the case is one of residual

poliomyelitis, though momentary consideration might be given to Friedreich's ataxia on account of the Babinski, pes cavus, absent knee and ankle jerk, scoliosis and questionable nystagmus. But the absence of familial history and chiefly the absence of ataxia speech disturbance, the stationary condition, the typical crossed paralysis, etc., rule out the hereditary disease.

The positive Babinski on the paralyzed side and the probable one on the non-paralyzed side speak of pyramidal tract involvement despite the preserved abdominals and cremasterics. Whether in this case we are dealing with an extensive cord lesion which has taken in the crossed and uncrossed pyramidal on the same side giving us the Babinskis, or whether there had been a concomitant encephalitis which now accounts for them, it is difficult to say. The fact that there is slight strabismus speaks for a possible polioencephalitis.

The kypho-scoliosis of which the patient complained very likely is the slow result of the weakened muscles of the spine unsuccessfully trying to keep the erect posture. The pain, too, may be attributed to the same cause.

SUBTEMPORAL DECOMPRESSION FOR ACROMEGALY; PRESENTATION OF A CASE

BY S. P. GOODHART, M.D.

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NEW YORK CITY

This patient is presented to the conference primarily for the purpose of demonstrating the value of subtemporal decompression in certain cases of acromegaly in which pain becomes persistent and intractable, and not amenable to more conservative measures. The case is of further interest in throwing some light upon the question, so often arising, as to whether we are dealing with hypersecretory activity of the pituitary gland without demonstrable hyperplasia, or with a true struma.

It involves furthermore consideration of the indications for either simple decompression or a procedure of a more radical nature, sella decompression, with or without effort at removal of soft tissue.

A discussion of the phases of the clinical picture will follow presentation of the case.

Patient, Paul M., male, age thirty-three.

The family and personal history throw no light upon the etiology of his present condition.

The patient's illness began about seven years ago (1911) with frontal and occipital pain, somewhat later radiating into both shoulders, and soon after occasional intense vertigo. Gradually the pain seemed to extend into the face, finally affecting the entire trigeminal area. This pain, more or less constant, was of such severe degree that the patient gave up his occupation. In February, 1915, there developed severe pain in the hands and in both knees; no macroscopic changes were then perceptible. Next described is a progressive general weakness and a noticeable enlargement of the facial skeleton. An intranasal local operation did not relieve the pain. The nature of and indication for this are undetermined.

Because of what was reported as positive serological findings patient received two courses of treatment for lues. Our examination at the Montefiore Hospital gave negative findings. The diagnosis of acromegaly and lues was positively made, however, at another hospital. During 1915 there were present polyuria and polydipsia. Libido and potency had also diminished during 1915. Upon admission to the Montefiore Hospital in November, 1915, patient's complaints were general weakness, very marked loss of weight—40 pounds within nine months, deep-seated occipital and frontal pains, pains in the hands and knees, polyuria, vertigo, and enlargement of the facial skeleton.

In December, 1915, there was likewise recorded an acromegalic hypertrophy of the phalanges. The thyroid gland now became enlarged. The pains in the head, frontal and occipital, and radiating into the face, continued most acute. This pain confined the patient absolutely to bed. Repeated examinations of the fields of vision and of the eye grounds revealed no pathological findings. A complete neurological examination made at various times gave no evidence of central involvement; nothing more was found than a slight difference in the activity of the knee jerks, the right more active than the left, and some asymmetry in the facial innervation, the right being somewhat less active than the left. In the latter part of 1915 it was observed by the examiner and confirmed by statement of the patient, that the hair growth had increased over the lower extremities. At this time the patient began to complain of radiating pains into the teeth with dysesthesias of the face and loosening of the teeth. A striking increase

of the activity of the sudoriferous glands was among the developing symptoms.

After specific treatment in May, 1915, neither in the blood nor in the spinal fluid were more positive findings determined than 1 plus Wassermann of the blood and of the cerebrospinal fluid, and an excess of globulin in the cerebrospinal fluid.

At various periods during observation at the Montefiore Hospital, varying quantities of sugar were found in the urine. For example, in November, 1915, there were nine-tenths of one per cent, somewhat later about one and a quarter per cent, and in January, 1916, as much as five per cent. It is to be observed that the patient usually responded to dietary treatment.

The intense continuous and exhausting pain in the head radiating as described, and lasting for months, made the patient absolutely bedridden; the slightest noise, a ray of light or slight pressure over the cranial or facial bones caused intense agony.

Glandular therapy was ineffectual. Thyroid gland very carefully given was seen even in small doses to increase the sugar excreted. Very careful experimental and therapeutic doses of pituitary gland were administered, but no definite results were obtained, and it is not possible for me to offer data of value as to opotherapy in this case. Our efforts to control the pain or to impress the growth of bone were unavailing. It was a question whether or not the patient should be given opiates *ad libitum*. There was manifestly not sufficient evidence upon which to base a diagnosis of pituitary tumor.

Spinal puncture showed no increase in pressure and gave no relief. Callosal puncture, sella decompression and subtemporal decompression were considered. The latter was determined upon. A right subtemporal decompression was performed by Dr. Elsberg on March 30, 1916. There was found some evidence of a pachymeningitis. The surgical recovery was rapid. For some few days after the operation the eye findings, especially as to the fields, were

variable. For example, three days after the operation, a left homonymous hemianopsia developed. A few days later the left pupil became dilated and responded only very slightly to light. There was a partial left ptosis with paresis of the left internal rectus. On the day following a bitemporal hemianopsia was determined. It is to be observed that these various findings were transitory. It is probable that an intraventricular edema of the third ventricle especially was the cause.

Some weeks after the operation the patient rapidly improved. The pain had entirely disappeared. Strength was regained; the return of the patient's mental alertness and general activity were striking. Entire relief from pain was in part responsible for this improvement. The bones of the skull and face were no longer sensitive to pressure: the glycosuria disappeared.

Our x-ray findings have perhaps been less variable than their interpretation. For example, in November, 1915, the radiographer reported greatly enlarged sella turcica; marked projection of the lower mandibula and of the basal portions of the frontal bone; thinning of the tabula externa, and to a less degree of the tabula interna of the basal portion of the frontal and anterior portions of parietal bones, and marked enlargement of the accessory sinuses of the nose as far as can be judged from the lateral view. Especially the frontal and sphenoidal sinuses showed an immense enlargement. (See accompanying x-ray prints.)

Skull.—Hyperaeration of the accessory sinuses of the nose more advanced. Otherwise condition practically the same as at previous examination.

Extremities.—The upper portions of the tibiae show a moderate amount of rarefying osteitis with beginning cystic degeneration.

Skull. Hands.—Compared with the previous plates taken before decompression, the condition of the bones of the hands and skull is practically the same on the present plate.

There is probably no doubt that in some

cases of acromegaly no morphologic or histologic changes in the pituitary gland can be demonstrated post mortem. In some cases which I have seen at the Montefiore Hospital with frank functional involvement of the gland, postmortem studies gave negative findings in the hypophysis. One case of this kind was especially instructive. We had this patient under observation for some time and obtained an autopsy. She was a girl of twelve. Without going into unnecessary details we may know that the clinical findings and the chemical and roentgenologic examinations warranted the diagnosis of an intracranial neoplasm, and pointed very strongly indeed to a tumor of the hypophysis. In addition to the general symptoms of brain tumor, a typical picture of the Fröhlich syndrome followed a short period of very rapid loss in weight. The patient had become totally blind from optic atrophy preceded by a very questionable papilledema; in fact the papilledema was denied by some of the observers, the optic atrophy was reported as primary, and from my examinations it so appeared, and was verified as such by the ophthalmologists. At the necropsy a tuberculoma involving the corpora albicantia was found. The pituitary was found morphologically and histologically intact; in the other endocrine glands no abnormal macroscopic or microscopic changes were found. The symptoms on the part of the pituitary were doubtless due to neighborhood pressure, perhaps edema, and the gland itself remained apparently structurally unimpaired. Undoubtedly, however, the function of the hypophysis had been very materially affected, at first reacting by irritation and by hyperfunction, and secondarily by hypofunction. It is perhaps a little presumptuous to declare absolutely that the hypophysis was normal even though a careful microscopic study was made. In a structure in which morphological, physiological and doubtless also chemical variation is so delicate and in which the process of preparation and examination

of the postmortem material is not at all uniform, one would show disregard for possible fallacies in observation to declare the gland normal; and regard for accuracy demands that one say no more than that with our present methods the pituitary showed no abnormal histological changes. The symptoms in this case were doubtless due to the proximity of the process within and involving the corpora albicantia at the base. At the Mt. Sinai Hospital the case was also regarded as probably of pituitary origin. The care with which one must interpret and give credence to roentgenologic plates in this region was exemplified in our case. The x-ray findings at different stages seemed variable, and yet it is hardly possible to suppose that such could have been the case; the bony envelope, the sella turcica and its osseous projections, could not have been appreciably affected, certainly could not have been returned to their normal relation after pathological enlargement; at autopsy the sella was found practically normal. Since observing this case, among others, with doubtful and variable roentgenologic interpretations, I have become sceptical of the absolute value of x-ray interpretation in new cases. I would only consider the plates of diagnostic value after repeated examinations and careful measurements of the enlarged sella.

In the case of acromegaly before us enlargement of the sella was reported early in the course of the symptoms, but as you will observe in the plate presented, taken about nine months after operation, the sella and its processes look quite within the normal. Indeed, even were there a true struma of the gland present, there should by this time have been definite developments. Only very recently has the patient again begun to complain of dysesthesia of the hands and cephalalgia suggestive of the same radiating character as at the onset. It would seem that only a series of roentgenological plates indicating progressive enlargement of the sella with thinning and absorption, should be accept-

ed as proof of a probably growing struma. In cases of hyperplasia of the gland, such as we commonly see resulting in acromegaly, the sella turcica remains structurally intact. Even with exacerbations, it is unusual for the gland to remain hyperplastic anatomically so that a true struma develops; in the case of the thyroid, however, the tissue, as we know, readily takes on a strumous character. Functional hyperplasia of the pituitary, I think we may definitely say, can occur without macroscopic and probably without permanent microscopic changes. Many of the characteristic subjective symptoms, such as headache of bitemporal character, radiating through one or more of the trigeminal branches, may be due to a temporary true hyperplasia of the whole gland with resulting distension of the sensitive dural envelop, or to a local edema. We must remember the likelihood of dysfunction of polyglandular character with most variable



FIG. 1.

manifestations, nor must we forget that a simple hyperplasia may develop into structural changes of a strumous nature. Under the stimulus of what Cushing calls a biochemical agent, the functionally unstable



FIG. 2.



FIG. 3.

gland may take on growth—adenomatous in type. As in the case before us, these changes are not so infrequent and it is just in such conditions as this that repeated x-ray and clinical examinations are of value in determining the nature of the process and consequently of the therapeutic

measures for relief. In the absence of objective evidence of a neoplasm or strumous growth within the sella, it would seem that for the relief of the persistent and agonizing pain the comparatively harmless surgical procedure of the subtemporal decompression should be utilized.

COMBINED SCLEROSIS LUEITICA

PREDOMINANT POSTERIOR COLUMN INVOLVEMENT OF TABETIC NATURE WITH BILATERAL PYRAMIDAL TRACT SIGNS

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The rather infrequent occurrence of combined sclerosis on a luetic basis warrants reporting this case. The patient, Mrs. R.L., fifty-four years of age, was born in the United States. Her chief complaints are: Weakness of legs; difficulty in walking; difficulty in starting urinary stream; pain in her hip, buttocks and thigh, and severe cramping of her toes.

HISTORY.—The onset of her symptoms dates back to about ten years ago, when she began to suffer from attacks of severe lancinating pains in the right leg. These attacks of pain would come on suddenly, without apparent provocation; they were sharp and stabbing in character, and would last from a few minutes or hours to the greater part of a day. At first the attacks were infrequent, about two or three a year; but gradually they came more frequently, until the patient suffered an attack every month or six weeks. Four years ago she began to have difficulty in walking; the loss of control of limbs was more marked on the right side than on the left, and has gradually been getting worse. Two years ago she began to suffer from pains in the lumbar region of the back; her toes would cramp up, and about this time she began to notice the loss of control of her bladder. For a time she became completely incontinent, but regained control of her urine about one year ago, since which time she has had difficulty in starting her urinary stream and some pain when urinating. She claims not to be able to tell when her bladder is full. She also suffers from marked constipation. Vision in her left eye

has been getting poor for some time. She never had any diplopia. Her appetite is fair, but she sleeps poorly.

Family History.—Her husband died of general paresis eleven years ago. She had one child who died from typhoid fever at the age of seventeen. She had one spontaneous miscarriage of a two months' pregnancy.

Past History.—Menstrual history normal. Menopause eight years ago. Patient never had any serious illness and was always robust.

Physical Examination showed a well nourished woman whose mucous membranes and general appearance were normal. Her heart showed slight hypertrophy and her second aortic sound was somewhat accentuated; there were no murmurs at any of the valves. Her lungs were normal.

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—There was a marked eversion of the right foot due to hypotonia; on the left side it was less marked but definite. There was a persistent dorsiflexion of the big toes of both feet, giving a constant Babinski attitude, the right side being more pronounced than the left.

Gait.—There was a well defined spastic ataxia gait which was very much worse when the patient walked with her eyes closed.

Coordination.—Standing with eyes open was fairly well done, but with eyes closed and when the feet were placed together, a well defined Romberg was elicited. Finger to finger and finger to nose tests were

accurately performed. Non-equilibratory ataxia was absent, but in the heel to knee test the range of movement was greater than normal.

Skilled Acts.—Handwriting was good and there was no defect in speech. Test phrases were repeated perfectly.

Abnormal Involuntary Movements.—There was no tremor either of tongue, facial muscles or hands.

Reflexes:

	Right	Left
Biceps	1	1
Triceps	1	1
Pectoral	1	1
Suprapatellar	3	3
Patellar	3	3
Achilles	1½	—1
Ciliospinal	0	0
Upper lat. abdom.	1	1
Lower lat. abdom.	1	1
Plantar	2	0
Babinski	Present questionable	
Oppenheim	"	"
Chaddock	"	"

Muscle Strength.—Rising from recumbent to sitting position was well performed.

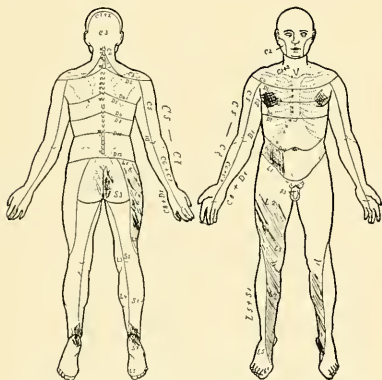


FIG. 1. SHOWING AREA OF ANESTHESIA TO TOUCH, PAIN, TEMPERATURE, POSTURAL AND VIBRATORY SENSES.

The muscle strength throughout the body was fairly well preserved. There was no definite atrophy of any of the muscles. The muscles were soft. There was a marked

diminution of tone in the muscles of both ankles, particularly on the left side; this hypotonus allowed complete dropping of the longitudinal arches of both feet with marked eversion. Hypotonus was also very well marked in the left arm. The thighs could be flexed on the abdomen to a considerably greater degree than normal. There were no abnormal associated movements.

GENERAL SENSORY. *Touch, localization and discrimination* were normal. Pin prick was diminished in areas as illustrated in chart. Temperature, muscle joint sensibility were fairly good. Pressure pain sense was moderately diminished over the lower extremities, and was especially well marked in tendo-achillis.

CRANIAL NERVES.—The olfactory nerves were not tested. Vision in the right eye was good; in the left eye, it was considerably diminished. The right fundus was normal; the left showed a slight chorioretinitis which the ophthalmologist reported as probably not luetic. The pupils were slightly irregular; the right reacted almost imperceptibly, while the left was absolutely fixed to light; both reacted to convergence and accommodation. There was no limitation to the eye movements, no ptosis, nor was there any nystagmus present. The other cranial nerves were normal. There was no subjective disturbance in hearing; objectively both right and left showed 20/20 hearing.

Her mental examination did not reveal any behavioristic abnormalities. Her responses to questions were prompt and correct; her intelligence was above that of the average individual; her emotional reactions were normal, her attention and calculation were good; her memory for recent past and remote events was good, and there were no hallucinations or delusions.

LABORATORY FINDINGS.—Examinations of her blood showed a 4 + positive

Wassermann; spinal fluid also 4 + positive with .8 c.c. and 2 + with .6 c.c. of fluid. Colloidal gold reaction showed a luetic curve. No estimate of the number of cells was made. Examination of the urine on four different specimens showed a specific gravity that varied between 10-14 and 10-17, a trace of albumin, no casts or sugar. Blood pressure was systolic 170, diastolic 100, giving a pulse pressure of 70 mm. of mercury.

SUMMARY

We have a patient whose husband died of general paresis eleven years ago; who began to have severe root pains ten years ago,

gradually becoming more frequent; with a disturbance in gait four years ago, that is progressively getting worse; disturbance of vesical control two years ago, with weakness and cramping of legs and toes.

PHYSICAL EXAMINATION showing a bilateral Argyll Robertson pupil; chorioretinitis; Romberg; hypotonia; diminution of Achilles reflex on both sides; with diminution of pressure-pain sense and areas of disturbed superficial pain sensibility; a bilateral Babinski, Oppenheim and Chaddock response with exceedingly active knee jerks; a positive blood and spinal fluid reaction. The signs and symptoms all point to an involvement of the posterior and lateral tracts of the cord on a luetic basis.

PAVOR NOCTURNUS AND ITS TREATMENT

BY ADOLPH STERN, M.D.

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NEW YORK CITY

The first patient I describe presents in addition to "night terrors" symptoms of insomnia and anxiety. I show the patient for two reasons; first, that he may tell you how he feels, and second, because I want you to see an individual who has had what one may call unusual sexual experiences, and yet remains a normally moral member of society. In psycho-analytical work, we find much in the lives of patients the narration of which gives the listener the impression that the patients are criminals or outcasts. The important point to remember is that as a rule only a person who strives against fulfilling wishes which are ethically wrong develops a neurosis. A defective individual yields to these impulses in a greater measure and becomes a criminal or a pervert.

J. B., thirteen years old, came to the Vanderbilt Clinic December 2, 1918, complaining that "for six years I am nervous when I go to bed at night. I see all sorts of things before going to bed that scare me in sleep and when awake."

The insomnia and night terrors prompted the boy to call for his mother, whose presence, after a light was struck, calmed him and induced sleep.

The phantasies which prevented sleep were of the following nature: Somebody carrying on his back a man all "slashed up," blood streaming from the wounds. Again somebody covered with a sheet, blood streaked. The dreams were equally terrifying; a typical one was the sight of Charles I being beheaded, the blood streaming from the body. Apprehension was produced in the daytime by the idea that someone was behind him, ready to do him harm. At other times he had a phantasy of someone standing at the head of the staircase of his apartment, and it was with great anxiety that he rushed past the object into his home.

The previous history of the patient shows a normal intellectual development, with a standing in school up to his grade. He is obedient, of a decidedly religious turn of mind, accustomed to a very strict, rather suppressive discipline in his home. On the whole, however, the patient is happy and contented and manifests a lively and healthy interest in both work and play.

The patient shows a very marked sexual precocity. At five years of age, influenced by larger boys, he indulged on several occasions with little girls in sexual practises of exhibitionism and handling of the genitals, and at the age of seven, attempted coitus with a girl of 15. He masturbated up to one and one-half years ago. For his offenses, he was severely beaten and threatened with being sent to jail and becoming a social outcast.

P. E. *Negative*.—There are many points to be considered in this patient. Under the conditions as they exist in an active dispensary, such as the Vanderbilt Clinic, those features of the situation which demanded most immediate treatment were first considered. They were the insomnia and night terrors.

While physical causes such as enlarged tonsils and adenoids, errors of diet and the like, and also psychic causes such as fright, have been mentioned as productive of night terrors and insomnia, psychoanalysts have repeatedly demonstrated that elements other than those above mentioned are etiological factors in a condition such as that from which this patient suffers. The active sexual manifestations present in the patient over so long a period, and their sudden cutting off (the stopping of masturbation one and a half years ago, following punishment and threat) are important considerations from the therapeutical point of view.

An incident not previously mentioned is that the patient has for many years slept in the same bed with his younger sister. Nothing of an improper nature, as far as I can ascertain, had taken place between the two. However, it is not unreasonable to assume that in the case of a boy sexually hyperesthetic, as our patient is, such close proximity to a girl might give rise to sexual stimulation, with consequent suppression of the desire, and anxiety as a result. Removal of the source of irritation is a logical procedure.

Another incident, previously mentioned, was that the boy in his fright at night always called his mother, whose presence comforted him. Freud has suggested that at least one of the sources of fear at night in children is that they miss the parent, a love object. However, a boy of thirteen is too old to feel the absence of a parent at night.

In view of the above two incidents, I advised the parents that the patient should sleep in a separate room, and that no one should respond to his cries at night, either in person or by voice, assuring them that in the course of a few days he would sleep without disturbance. I told the patient the same.

In addition, on several occasions I discussed with the boy his sexual difficulties. The only medication given was 3 ss R & S at night. At the end of a week the boy slept soundly and since then has not suffered either from insomnia or night terrors.

Other aspects of this patient present problems which may or may not solve themselves with time. A difficulty which the boy himself recognized was the readiness with which he was roused to intense anger. On such occasions, as he puts it, "I feel terribly strong, and could kill any man." However, he manages to restrain himself. His dreams, as for instance the beheading of Charles I., represent but thinly veiled though wholly unconscious hostile wishes directed towards the father. The before-mentioned inclination to intense

anger probably has as one of its determinants the same unconscious root, i. e., the father rivalry. It is also generally held by psycho-analysts that the phenomenon of the night terrors, especially the call for the presence of the mother, has as its unconscious cause the mother complex.

It is interesting to note that a form of day phantasy frequently indulged in by the patient consisted of intensely ambitious schemes whereby the boy made great fortunes and kept the family in affluence. This may be regarded as in line with the above, in which the patient has replaced the father and is now taking care of the family. This is a very common form of day phantasy in neurotic boys and I might add in adults also.

These are phases of developmental life which many individuals have gone through successfully, and it may easily be that with the explanation the patient obtained at the dispensary he will continue to do well.

The second patient that I report has not returned to the dispensary since her first visit. I therefore am not in a position to state the results of the procedures instituted. In view, however, of the fairly uniform success in properly selected cases, I trust that here, too, we have succeeded, and shall report this patient.

N. P., a girl of seven, the older of two children, came to the Vanderbilt Clinic Feb. 1, 1919, having suffered for the past six months from insomnia and night terrors. In the summer of 1918, the patient was awakened one night by hearing loud talking in the street, and since then has awakened every night with fear and found it difficult to fall asleep afterwards.

The mother has noted some other significant changes in the patient, namely, since the onset of the illness, she has become more childish, cries more readily, clings constantly to the mother, following her from room to room. In general the child is well behaved and obedient, but since its illness tends to do things of a minor nature to irritate the mother.

When the child awakes in a terror she sits up, opens her eyes wide, cries out, "There is the man: there he is," and with a motion of her arm, pushes the disturbing object aside. She recalls nothing of this afterwards. She sleeps only if she is taken into the bed of her parents or if her bed is drawn quite close to that of the parents. Moreover, the child had always slept in the same room with the parents except for one week in October, 1918, when she slept in a separate room. During this time she slept well. Circumstances then necessitated a return to the previous sleeping quarters, with a return of the symptoms.

Otherwise the child's history is perfectly negative, as is also the result of a physical examination.

Psychoanalytical work in adults has repeatedly demonstrated that parents or others in charge of children do not give sufficient heed to the powers of observation of little ones or realize to what an extent the latter weave into their lives, often for harm, their own interpretation of what they see and hear in the bedroom of their

elders. I believe that this has happened in the case of our patient, namely, that she saw the parents in some act which aroused fear (or curiosity) and that this manifested itself in dreams of attack by a man of whom she freed herself by awaking.

The apparently recently manifested attachment to the mother, whom she now purposely irritates, indicates a desire by day to be with the parent similar to that fulfilled at night through the pavor nocturnus, for as you recall, the child was taken into the parents' bed in order that she might fall asleep.

In this case also I advised the parents to separate the sleeping room of the child from their own, and not to respond by their presence or voice to her calls. I also explained to the patient what would be done and assured her that though she may continue to wake for several nights, she would cease to do so after that. Medication consisted in giving a placebo. As I stated before, I cannot report on the success or failure of the methods used in the case of this patient.

POST-INFLUENZAL PARALYSIS OF THE PERIPHERAL MOTOR NERVE TYPE *

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The recent influenza epidemic, besides the usual pulmonary involvement, in a large number of cases has not spared the cerebro-spinal system. All types of nervous involvement were observed, and afforded an excellent opportunity for study. The clinical picture in some of the cases was readily interpreted, while in others the bizarre neurologic phenomena were indeed quite puzzling. The case here reported simulated somewhat the paraplegic type of anterior poliomyelitis, and probably would have been considered such at any other time. Its association, however, with an attack of influenza, and the steady and rapid improvement, greatly facilitated our arriving at a proper conclusion.

The patient is a girl 15 years of age, of Italian parentage, who came under observation complaining of weakness in the legs and knees dating from an attack of influenza in October, 1918, the severity of which necessitated a two months' stay in bed. At the end of that time on attempting to get out of bed her knees suddenly gave way and she would have fallen but for the timely help of her mother. Thereafter she was able to get about only with great difficulty, even when assisted by several persons. This state of affairs continued up to two weeks ago when she was able to walk alone, not however without considerable difficulty. At no time during her period of illness did she complain of spontaneous pain in the limbs. Pain could readily be elicited, however, by pressure on the muscles, or contact with neighboring objects.

Her history prior to the attack of influenza is without importance. Three other members of the family who were ill

at the same time recovered without any sequelæ.

EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Abnormal attitudes and deformities.*—None noted.

Gait.—This is markedly impaired, the steppage type of gait being quite in evidence. There is considerable unsteadiness and trepidation which can be readily explained by the impairment of the muscular power of the extremities.

Coordination.—No abnormality noted in the coordinative acts of the upper extremities. Some impairment noted in the lower extremities but this can readily be explained by the lack of motor power in the muscles of the thighs and legs.

Dysmetria.—None noted.

Skilled acts.—Performed in a normal manner.

Abnormal involuntary movements.—None noted.

Muscle strength.—No impairment in muscles of trunk, upper extremities and neck. Decided weakness in all types of muscular efforts in thighs, legs and feet.

Muscle status.—All the muscles of the lower extremities show a moderate degree of atrophy. Their consistency is soft and there is marked loss of tone. Vasomotor disturbances are noted, such as cyanosis and coldness of the affected muscle groups.

Electrical reactions.—Complete R.D. in all the muscles (flexors and extensors) of the thighs and legs. Faradic response not obtained and polar inversion to galvanic current present.

Reflexes.—All deep reflexes for upper extremities present and equal. All deep reflexes for lower extremities completely

*From the service of Dr. Henry W. Frauenthal. Presented at Clinical Conference February 24, 1919.

absent. No ankle clonus or pathological reflexes present. All superficial reflexes present and equal on both sides.

GENERAL SENSORY.—Slight hypesthesia and hypalgesia in both lower extremities, but this was not constant. At times, thermic and sharp point stimuli were interpreted as hyperesthetic, at other times they appeared to be normal. There were no spontaneous dysesthesias.

CRANIAL NERVES. *Olfactory and nose.*—Normal.

2nd and optic.—Normal. Pupils react promptly to light and accommodation.

Oculomotor apparatus.—Show no abnormalities.

3rd, 4th and 6th.—No nystagmus present.

5th and trigeminus.—Normal.

7th and facial.—Normal.

8th and acoustic.—Normal.

9th and glossopharyngeal.—Normal.

10th and vagus.—Normal.

11th and spinal accessory.—Normal.

12th and hypoglossus.—Normal.

MENTAL STATUS.—Excellent.

SYSTEMIC EXAMINATION.—Reveals no noteworthy abnormalities.

SUMMARY AND DIAGNOSIS

In considering this case in all its aspects, very little difficulty should be experienced in arriving at a diagnosis. The attack of influenza and the subsequent development of the paralysis readily incriminated the toxin of that disease as the cause of the neuritic disturbance, for we are well aware of the destructive action influenza has on nervous structures. No part has escaped its ravages. The cortex, the meninges, the nuclei of the midbrain and bulb have all felt the effects of the invader. In the case presented the peripheral motor nerves are obviously involved. The symmetrical character of the paralysis, the areflexia, the atrophy and atony, and the R.D., unerringly point to a neuritic involvement of the peripheral motor nerves.

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CONJUGATE DEVIATION OF THE EYES WITH A PRESENTATION OF A CASE

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A consideration of the subject of conjugate deviation of the eyes is of interest both from the clinical and the anatomical points of view. The subject has been given a great deal of study and investigation by the French students of neurology and as a result, mainly of the work of M. and Mme. Déjerine, we are able to separate the manifestations of disturbed associated movements of the eyeballs into a number of types and groups. This classification rests on the evidence of both clinical and anatomical data.

The cortical center, to which has been attributed the voluntary control of conjugated movement of the eyes, is located in the posterior portion of the second frontal convolution. In this region are also located the cells whose axones control the lateral movements of the head and neck, and we shall later see that these two functions are closely related and that the fibers which subserve both functions lie in anatomical proximity in their course through the internal capsule, crus cerebri and pontine regions. The axones which convey the impulses destined for the conjugated movements of the eyes are called *oculo-gyric fibers* and the impulses passing over them, the *oculo-gyric impulses*. Likewise those fibers which conduct the impulses for the lateral movements of the head and neck are called *cephalo-gyric* and the impulses producing such movements, the *cephalo-gyric impulses*. The

combined movements of the eyes and head which may arise either reflexly, automatically or voluntarily are called *oculo-cephalo-gyric movements* and the impulses which produce such movements, the *oculo-cephalo-gyric impulses*.

The axones of the cortical cells which pass to the nuclei of the cranial nerves are not incorporated with the main mass of the pyramidal tract axones but are anatomically separated from the latter tract throughout their course, at some levels being relatively distant from the pyramidal tract, at other levels adjacent, but at all times the two systems are distinct. These axones form the so-called cortico-nuclear or pallio-nuclear connections and bring the cranial nerves under the control of the cerebral cortex. The collections of nerve fibers destined to pass to individual or correlated cranial nerve nuclei are called the *aberrant pyramidal* fibers inasmuch as they leave their neighbor, the pyramidal tract, at varying levels to enter upon new and unexpected pathways in their course to the cranial nerve nuclei. In contrast to these cortico-nuclear fibers are the cortico- or pallio-spinal fibers which are destined to establish connections with the anterior horn cells of the spinal cord and which make up the crossed and direct pyramidal tracts.

In addition to the frontal area described as giving origin to oculo-gyric movements, other centers have been postulated as

existing in the parietal and occipital cortex, and some evidence will be adduced in the consideration of the case serving as the basis for this paper in support of an occipital center.

The axones of the frontal (voluntary motor) oculo-gyric cells arising in the gray matter of the second frontal convolution pass as projection fibers from their cells of origin into the white matter of the cerebral hemispheres and form part of the centrum ovale of the hemisphere. They converge toward the internal capsule and joining the other cortico-nuclear fibers, pass into the compartment in the internal capsule which is located at the knee of the capsule immediately anterior to the compartment occupied by the main pyramidal mass of fibers.

Immediately upon emerging from the inferior or ventro-caudal aspect of the internal capsule, we find that the dispersion of this cortico-nuclear fiber tract begins, and at varying levels collections of fibers leave the tract and, passing in great part across the median line, supply the nuclei of the cranial nerves. A small number of fibers remain uncrossed and supply the nucleus of the same side, thus providing a double innervation from both sides of the cortex.

After emerging from the internal capsule, the cortico-nuclear fibers enter the crus cerebri lying mesial to the main mass of the pyramidal tract. The tract becomes smaller and smaller through the defection of collections of fibers passing to the cranial nerve nuclei as we pass caudally, until it ceases to exist and the last survivors swing dorsally as the contingent destined to supply the hypoglossal nuclei, at a level corresponding to the junction of the pons and medulla. In the pons, the tract continues in the same mesial relationship to the pyramidal tract observed in the mesencephalic region.

The oculo-gyric contingents of the aberrant pyramidal fibers are three in number, the hypothalamic bundle, the pes lemniscus superficialis and the pes lemniscus

profundus. The hypothalamic aberrant pyramidal fibers leave the cortico-nuclear tract as it emerges from the internal capsule just ventral to the thalamus; passing dorsally, they descend for a short distance in the mesial portion of the mesial fillet, the great sensory fiber tract from the medulla to the thalamus, and then swinging dorsally, they end in the nuclei of the III and IV cranial nerves, the great majority of the fibers passing across the mid-line, a few remaining on the same side. The next contingent, the pes lemniscus superficialis, leaves in two strands at the upper third of the crus cerebri, joins the fillet and descends for a short distance, reaching a level corresponding to the caudal limit of the superior colliculus where a portion of it swings dorsally and ends in the III and IV cranial nerve nuclei, the remainder passing downward to supply the VI cranial nerves. The third contingent, the pes lemniscus profundus, contains the fibers which conduct the impulses for the oculo-gyric and cephalo-gyric movements. This bundle leaves the cortico-nuclear tract at the level of the sulcus between the crus cerebri and the pons, joins the mesial fillet, descends to the level of the VI cranial nerve nucleus where a bundle of fibers passes dorsally to the sixth nucleus. The remainder carrying cephalo-gyric movements continues to descend in the fillet and eventually supplies the cell groups governing the lateral movements of the head and neck, in large part the supply of the sterno-cleido-mastoid and the trapezius muscles.

This, in general, is the course of the oculo-gyric fibers from the cortex to the nuclei of the III, IV, and VI cranial nerves. The maintenance of the integrity of the conjugate movements of the eyes depends upon the continued normal function of this cortico-nuclear pathway. In addition to this, the coordinated movements of the eyes depend upon the integrity of the nuclei and the emergent fibers of the III, IV and VI cranial nerves and to a number of other additional factors,

the consideration of which we can now discuss.

The three oculo-motor nuclei are brought into intimate relationship by a strand of coordinating fibers which lies in the posterior portion of the tegmentum of the midbrain and pons in close proximity to the nuclei of the III, IV and VI cranial nerves. This bundle is called the *posterior longitudinal fasciculus* and the relationship between these three nuclei conditioned by this bundle of fibers renders possible the coordinated movements of the two eyes. The aberrant pyramidal fibers going to the VI nerve nucleus and the nucleus itself not only govern the external rectus of the eye on the same side of the body, but have almost complete control of the internal rectus of the eye on the opposite side of the body.

The abducens nucleus has been called the pace-maker of the ocular movements and its control is exerted over the external rectus of the opposite side by decussating fibers and the III and IV nuclei through the posterior longitudinal fasciculus. In addition, we find fibers reaching the VI nucleus and the posterior longitudinal fasciculus from the deiteral complex of the vestibular nerve which exert an influence upon the oculo-gyric nuclei and, through them, upon the conjugate movements of the two eyes.

The situation of lesions which may interfere with conjugate movements of the eyes may be catalogued and located as follows:

1. *The Cerebral Cortex*—the foot of the second frontal convolution.—A lesion in this locality or in the immediately sub-jacent white matter will produce disturbances in the oculo- and cephalogyric movements, both being involved.

2. *The Internal Capsule*.—Here the lesion will produce disturbances in the entire cortico-nuclear group of fibers and disturbances would be noted in the innervation of the masticatory, the facial, the pharyngeal, laryngeal and the lingual musculature. In all probability there would

be, in addition, disturbances of the pyramidal tract itself with the signs and symptoms of a hemiplegia varying in intensity with the degree of disturbance in the tract.

3. *The Hypothalamic Region*.—In this locality we find a syndrome which has received the name "vertical paralysis of gaze" from the fact that the lesion involves

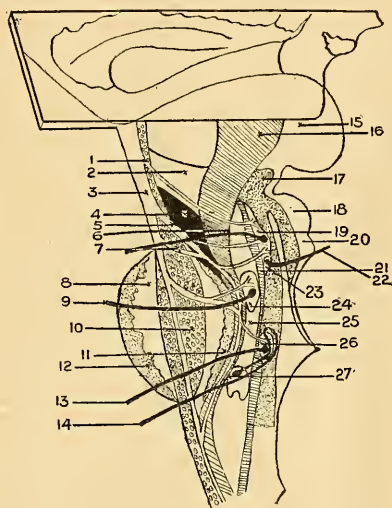


FIG. 1. COURSE OF THE OCULO-GYRIC CONTINGENTS OF THE ABERRANT PYRAMIDAL TRACT (AFTER DÉJERINE).

- | | |
|-----------------------------|--|
| 1. Hypothalamic Bundle. | 15. Thalamus. |
| 2. Reticular Formation. | 16. Mesial Fillet. |
| 3. Cortico-nuclear Bundle. | 17. Central Gray. |
| 4. Substantia Nigra. | 18. Superior Colliculus. |
| 5. Pes Lemniscus Superf. | 19. Third Nerve Nucleus. |
| 6. Third Nerve. | 20. Inferior Colliculus. |
| 7. Pes Lemniscus Profundus. | 21. Posterior Longitudinal Fasciculus. |
| 8. Pontine Nuclei. | 22. Fourth Nerve. |
| 9. Fifth Nerve. | 23. Fourth Nerve Nucleus. |
| 10. Pyramidal Tract. | 24. Fifth Nerve Nucleus. |
| 11. Dorsal Pontine Fibers. | 25. Reticular Formation. |
| 12. Ventral Pontine Fibers. | 26. Sixth Nerve Nucleus. |
| 13. Sixth Nerve. | 27. Seventh Nerve Nucleus. |
| 14. Seventh Nerve. | |

the aberrant pyramidal bundles destined to supply the III cranial nerve nucleus. This usually manifests itself in a bilateral

paresis or paralysis of the superior and inferior recti muscles, together with a disturbance in the associated movements of the superior oblique muscle, producing an inability to raise or lower the eyes, movements to the right or left remaining completely free from disturbance.

4. *The Crural and Ponto-crural Region.*—

In this region we may find an involvement of the conjugate movements of the eyes to one or the other side, alone or in combination with a disturbance in movements of the head and neck. In the former case, the pes lemniscus superficialis, most of whose fibers are oculo-gyric, is disturbed; in the latter case, the lesion probably affects the pes lemniscus profundus, part of whose fibers are oculo-gyric, and part cephalo-gyric. In either case, lateral movements of the eyes to the right or left will be affected through a disturbance of the VI cranial nerve, the pace-maker. A lesion of the pes lemniscus superficialis is more apt to give a disturbance of the conjugated lateral movements of the eyes without other disturbances, whereas a lesion of the pes lemniscus profundus must anatomically occur at or near the crural-pontine sulcus, and in that case there will be present symptoms due to disturbance in the facial nerve which lies in close relationship. A lesion of the VI nerve nucleus itself will in all certainty give a disturbed facial innervation, so that absence of a facial paresis or paralysis will necessitate the elimination of the pes lemniscus profundus or the VI nerve nucleus itself as the locus of disturbance.

These disturbances of conjugate movements of lateral gaze were first described by Foville and they bear his name, the *syndromes of Foville*.

5. *The Posterior Longitudinal Fasciculus.*

—A lesion of this connecting pathway between the oculo-motor nuclei will give disturbances in lateral gaze, but in this case also the contiguity of the facial nerve in its internal course will supply a localizing means of diagnosis. Failure to find disturbance in the facial nerve will render

improbable a lesion in the posterior longitudinal fasciculus.

6. *The Deitero-oculo-motor Connection.*—

Interference with this connection will produce disturbances in lateral gaze but they will be associated with other equilibrium phenomena and nystagmus.

Disturbances in the oculo-gyric pathway between the thalamus and the pons will usually be associated with disturbances in sensation, on account of the anatomical relationship between the oculo-gyric fibers and the median fillet, the thalamo-medullary link in the great sensory pathway from the periphery to the post-central convolution.

As Head has pointed out of late, a gradual rearrangement of the fibers of the median fillet takes place between the medullary nuclei of Goll and Burdach and the nuclei of the thalamus, whereby a change in the integration of sensation occurs—the discriminative judgment components tending to be collected together, and the effective components drawing closer to each other. This becomes more and more complete the nearer we approach the thalamus, and becomes complete in the final rearrangement in the thalamic nuclei from which the fibers pass to the cortex.

Motor disturbances are not of marked degree unless the lesion is a wide-spread one, for both the pyramidal and the extra-pyramidal systems are located at some distance from the fillet and its contained descending cortico-nuclear fibers.

I. LESIONS AFFECTING VOLUNTARY MOTOR CONTROL.

1. *If the lesion is irritative:*

- (a) If the location is supranuclear in position, the eyes will be drawn away from the side of the lesion.
- (b) If the location is nuclear or infranuclear, the opposite will be the case and the eyes will be directed toward the side of the lesion.

2. *If the lesion is paralytic:*

- (a) If the location is supranuclear in position, the eyes will be drawn toward the side of the lesion through the unopposed action of the antagonists of the paralyzed muscles.
- (b) If the location is nuclear or infranuclear, the eyes will be directed away from the side of the lesion through the same mechanism.

II. LESIONS AFFECTING THE STABILIZING CONTROL through an interference with the vestibular oculo-gyric pathway on one side of the pons will produce an involvement of the eye movements on that side associated with nystagmus.

- (a) An irritative lesion will produce a deviation toward the side of the lesion.
- (b) A paralytic lesion will produce a deviation away from the side of the lesion.

III. LESIONS AFFECTING THE INTERSEGMENTAL ASSOCIATION CONTROL through the posterior longitudinal fasciculus will serve in the main to destroy the conjugated movements of the eyes without producing any definite conjugate deviation. If the connecting link between the abducens and the oculomotorius nuclei be destroyed, we would then find a dissociation in the coordinated movements of the eyes; impulses which would previously have produced a conjugate movement to the right, for example, will still produce a movement of the right eye to the right, but the left eye will fail to move to the right also through a failure of contraction of the internal rectus muscle of the other eye.

THE CASE

F. B., age thirty-three, single, Italian, barber, came to the Vanderbilt Clinic and examination was made on April 9, 1919.

Previous History.—His habits are good; he does not use alcohol or drugs of any kind. His appetite is good and he is gaining in weight. He has never had any head injuries. In 1903 he fractured his right leg below the knee and in 1914 he had an operation for hernia. He has had gonorrhea many times, is subject to colds, but he has not suffered from any other illnesses.

Family History is negative.

Present Illness.—The onset of his illness has been gradual. Three or four months ago, he noticed that the sensation of the fingers of the right hand was disturbed; the hand felt as if he were wearing a glove. This condition improved somewhat when he stopped work, but of late it has returned and become worse. He thinks that the power of the right side is just about as good as it was formerly but he complains of general weakness. He states also that there is a constant ringing in the right ear. There is no trouble with the left arm and leg. He volunteers the information that he has lost the ability to recognize objects by palpation; he is aware of the fact that there is something in his hand but he has no idea of the nature of the object. He does not know whether his ability to recognize pain and temperature has been impaired. There has not been any form of tremor or twitching on the right side. One month ago, he noticed that he saw double and at that time the right eye was drawn over toward the right; about one week later, the left eye became affected, also turning toward the right. He took some medicine and for about two weeks after this, he had some trouble in speaking, which has since cleared up. There is no disturbance in vision except that incident to the deviation of the eyes to the right side. He complains of dizziness but has no headache. There have been no attacks of falling and no convulsions. He had a heavy cold in January, was sick in bed with a fever, but did not have a physician. His condition is gradually improving.

EXAMINATION

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes.—He sits with his head turned toward the left and his eyes turned into the right canthi, more or less fixed.

Gait.—He is weak and unsteady when walking on a line, on his toes or heels. Fournier's tests are positive—walking is more difficult when he closes his eyes.

Coordination. Equilibratory.—This is normal when standing on the left leg but difficult when standing on the right leg, especially when the eyes are closed.

Non-equilibratory.—There is some ataxia and tremor of the intention type, more marked in the right arm. The finger-nose and finger-finger tests are done with much difficulty, not perfectly performed by the left arm. There is no definite past-pointing. The test to bring the toe to an object above the recumbent position is well done with the left foot but is difficult with the right. The heel-knee test is better performed with the left than with the right foot. There is no dysmetria and no adiadochokinesis.

Skilled Acts.—He has some difficulty in snapping the fingers and in approximating the thumb and fingers. Even with the left hand this is not done perfectly with the eyes shut. A specimen of his handwriting shows ataxia and awkwardness. The speech is normal.

Abnormal Involuntary Movements.—There are no abnormal involuntary movements except for the slight tremor mentioned above.

Reflexes.—

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Jaw	2	2
Pectoral	1 pl.	1 pl.
Biceps	1-2	1-2
Triceps	1-2	1-2
Radial	1-2	1-2
Ulnar	1 pl.	1 pl.
Wrist	1 pl.	1 pl.
Suprapatellar	1-2	0-1
Patellar	2-3	2
Achilles	2	2

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Dorso-cuboidal	0	0
Periosteal uncrossed	0-1	0
Periosteal crossed, R. to L. o L. to R. 0-1		
No crossed patellar; adductor R-L.; slight L-R.		

<i>Superficial</i>	<i>Right</i>	<i>Left</i>
Ciliospinal	pr.	pr.
Supra-umbilical	0	0
Suprapubic	very sl.	very sl.
Upper lat. abdom.	0-1	0-?
Lower lat. abdom.	very sl.	Incon.
Cremasteric.	pr.	Incon.
Plantar flexion	1	2
Babinski	1	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schafer	0	0

Muscle Strength.—The right leg is slightly less strong than the left, but this is attributed to injury in youth.

Unopposed and opposed movements are normal except for the weakness noted above and a slight weakness in abduction of the right hand and pronation of the left arm.

Muscle Status.—The volume and contour of the muscles are normal except in the two lower extremities; the right leg in the midcalf region is 27 cm. and the left leg in the same region is 29 cm. The tone of the muscles is equal in the upper and lower extremities.

There are no *abnormal associated movements*.

GENERAL SENSORY. *Touch* is less acute over the entire right side, especially in the distal parts. *Touch localization* is about normal except on the lower extremity where power to localize is not so good. *Touch Discrimination.*—Two points are not recognized as such although separated more than twice as far on the right as on the left.

Pain is diminished over the entire right side.

Temperature.—Cold and warm are always distinguished as such but sensation

is not so acute on the right as on the left side, and more so in the leg.

Vibratory sensation is slightly reduced in the right upper and lower extremities.

Pressure sensation is unchanged.

Muscle-tendon sense is subjectively less marked on the right side, especially in the right toe.

Stereognostic sense is almost completely lost in the right hand, perfect in the left hand.

Barognostic sense is absent for moderate differences.

CRANIAL NERVES. *Olfactory Nerve and Nose.*—Normal.

Optic, Oculomotor, Trochlear and Abducens Nerves and Optic Apparatus.—Vision: acuity is normal; the fields are normal. The fundi show no pallor of the discs; the vessels and media are normal. The pupils are slightly irregular in shape, central in position, the right being 3 millimeters and the left 4 millimeters in diameter. There is a slight hippus. Their reaction to light direct and consensual and convergence are normal. There is no Argyll-Robertson pupil. The eyes show a conjugate deviation to the right. The movements of the eyes are fair upward, none inward, very slight downward and outward, to the right good, to the left both poor R/L. There are slight nystagmoid movements in all directions. Accommodation is good.

Acoustic Nerve and Ear.—Subjective noises in the right ear; objective acuity about equal in both ears. Rinné A/B, B referred to R.

Trigeminus Nerve and Mouth.—Motor movements to the right are poor; he bites hard on the right and left but the patient says he can bite better with the left side. Conjunctival reflex is normal. There are no abnormalities of the mouth.

Facial Nerve and Face are normal.

Glossopharyngeus and Vagus Nerves, Pharynx and Larynx are normal in all respects.

Hypoglossus Nerve and Tongue.—He cannot move the tongue quite as well as formerly.

SYSTEMIC.—The skin is scaly and rough. He has a left inguinal hernia. No glands are felt. His pulse is 80, full and regular, his blood pressure is 114 systolic and 70 diastolic. The heart sounds and outline are normal. His respiration is normal, gastro-intestinal system is normal, genito-urinary system is normal, and skeletal system is normal.

LABORATORY EXAMINATION

The Wassermann reaction in both the blood and spinal fluid is negative. There are 3 lymphocytes to the cubic millimeter of the spinal fluid. The Noguchi-butyric acid reaction shows a three plus reading. The colloidal gold curve is 1122100000.

April 11. The patient was seen by Dr. Strong who found an incomplete ophthalmoplegia, there being a beginning restoration of movement upward and downward. It was also found that if the patient fixed his gaze on an object and the head was rotated away from the object, the eyes would move somewhat toward the left, although there was no voluntary ability to turn the eyes toward the left. The sensory findings were unchanged.

April 14. Still further improvement in the eye movements. With rotation of the head, the eyes will now move to a point slightly beyond the mid-line toward the left. There are no sensory changes. The patient feels much better.

April 15. The eyes can now be made to move almost into the left canthi by moving the head. No sensory changes.

DISCUSSION OF THE CASE

The slight involvement of the right motor system and the distinct impairment of the right sensory system will localize the lesion on the left side of the brain since both motor and sensory distribution is of hemisomatic type and such lesions are almost invariably located above the motor and sensory decussations. Having located the side of the lesion, it now remains to determine its level. Since the disturbance

is hemisomatic, the level will be above the medullary region. The sensory changes will now serve to help us to a certain extent. The disturbance of sensation is confined in great part to the discriminative forms of sensation, those types of sensation which lead us directly toward judgment. This guide will lead us to place the lesion as high in the central nervous system as is compatible with our other findings. As Head of England has pointed out, these sensory disturbances are of the cortical, thalamic or hypothalamic type, for as the median fillet approaches its relay point in the thalamus, those fibers which carry the purely affective types of sensation begin to draw apart from the fibers which carry the discriminative types of sensation and small lesions will tend to affect one or the other but not both sets of fibers. This would lead us to place the lesion as near the thalamic region as possible.

The involvement of gaze can now occupy our attention. A consideration of this feature of the case will indicate a lesion of the cortico-nuclear path from the cortex to the nuclei of the oculomotor nerves. We can immediately eliminate the lower Foville syndrome, that of the pons, on account of the fact that the lesion does not affect the VII cranial nerve, for it is inconceivable that a lesion, no matter how exact, could involve the nucleus of the VI cranial nerve without a simultaneous involvement of the VII nerve. The character of the sensory changes would also indicate a lesion higher in location than the pontine region. This, then, will throw the lesion back on the supranuclear course of the oculo-gyric fibers passing to the VI cranial nerve. The absence of any involvement of the cephalo-gyric fibers will eliminate the cortex and centrum ovale as well as the pes lemniscus profundus. The fact that lateral gaze is involved places the lesion below the hypothalamic aberrant pyramidal contingent. By this process of elimination, we have arrived at an area stretching from the upper border of the superior colliculus above to the inferior colliculus below and in

this region we find the pes lemniscus superficialis passing downward within the median fillet, in which the process of rearrangement into judgment faculties is taking place. The sensory disturbance definitely places the lesion on the left side, and since the eyes are turned to the right, the lesion must be an irritative one which is involving the oculo-gyric fibers destined for the right external rectus and the left internal rectus.

Considerable discussion has been aroused by the fact that although the patient was unable voluntarily to bring the eyes beyond the mid-line toward the left, yet when his gaze was fixed upon an object and the head rotated toward the right the eyes swung toward the left and passed almost into the left canthi. This fact has been noted by previous investigators and has been promulgated as evidence that the lesion in such cases was supranuclear in character and location, which would accord well with our conclusions. The exact mechanism is, however, obscure and it is at least susceptible of explanation on other grounds. If we analyze our voluntary ocular movements, we can quickly conclude that these movements depend upon other impulses than sight; if at command we move our eyes to the right or left, it is not because we have seen something to the right or left but because we wish to see something in either of those directions; likewise auditory, tactile and olfactory stimuli lead us to try to bring the source of the stimulation into the field of macular vision. In this present case, however, we have to begin with a stimulated calcarine visual cortex which perceives the source of the stimulation in the center of the field of vision. Now as the head is rotated away from the object, the point of stimulation would tend to pass out of the line of visual fixation. This is contrary to ordinary experience, for once the attention is directed toward some object, the tendency is to retain the object in the plane of visual fixation. Anyone can subjectively test this by staring at a moving object

and the eyes will be found to follow this object without any voluntary effort. This, perhaps, may predicate other pathways than the ordinary one of voluntary control of the movements of the eyes, which may arise in the calcarine cortex and pass out in the occipital projection system, becoming linked up in the stem with the oculomotor nuclei. We can adduce no definite anatomical proof of the latter part of this contention, but it is at least worthy of consideration at this time.

A third element which can be considered now is the pupillary change. It is questionable whether this is of importance, but we may attempt to bring it into agreement with our other findings. In the midbrain at the level of the oculomotor nucleus, we find the emergent fibers of the III cranial nerve sweeping around ventrally through the red nucleus and passing through the mesial portion of the mesial fillet to gain their point of emergence from the brain stem, the sulcus oculomotorius on the mesial aspect of the crus cerebri. As they pass through the mesial portion of the fillet, they come into relation with that portion of the fillet which is furthest removed from the affective elements of the fillet, the spino-thalamic tract which has been caught on the lateral horn of the fillet lower down and which has been progressively shifted laterally by the migration of the fillet. In this region are also situated the aberrant pyramidal fibers destined for the right abducens nucleus.

Thus, although we have been forced to make certain allowances in the explanation of the symptoms exhibited in the case, we are drawn to the conclusion that the patient has suffered from a more or less evanescent and temporary lesion, probably vascular in character, which has affected the brain stem in the ventral tegmental region of the midbrain, producing a partial interruption in the judg-

ment or discriminative types of sensation, an irritation of the dextro-gyric aberrant pyramidal tract, and involving to a very limited degree the emergent fibers of the III cranial nerve sufficiently to produce demonstrable changes in the innervation of the sphincter iridis muscle. The predominating conjugate deviation may be sufficient to obscure other partial disturbances of the left oculomotorius nerve. The rapid amelioration of the symptoms during the week of observation will also tend to confirm this diagnosis.

The clinical diagnosis is as follows: Right astereognosis with conjugate deviation of the eyes to the right.

The anatomical diagnosis is as follows: A lesion involving the mesial fillet in its internal portion, together with the pes lemniscus superficialis contained within the fillet and the emergent fibers of the oculomotor nerve of the left side as it passes through this part of the mesial fillet.

The pathological diagnosis is as follows: A probable vascular accident, either thrombotic or hemorrhagic in character, which is in course of resolution.

In view of the foregoing facts, it has seemed an open question whether the etiological factor in this condition was epidemic encephalitis. The prevalence of midbrain lesions as demonstrated at autopsy in this disease, the absence of evidence of syphilitic infections in this case, the evanescent character of the clinical picture and the presence of an epidemic of the disease at the time of onset, would perhaps lead us to make a diagnosis of epidemic encephalitis. The character of the spinal fluid might be that characterizing epidemic encephalitis after the acute period had subsided.

We take this opportunity of expressing our appreciation to Professor Arnold H. Knapp for having referred the present case to us for study and publication.

THE THALAMIC SYNDROME IN EPIDEMIC ENCEPHALITIS

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The findings in this case of epidemic encephalitis are recorded in detail, as they point to an involvement of the optic thalamus and are of interest because of the clinical infrequency of lesions in this area. The photographs were taken to

illustrate the general position of the patient in bed and also to illustrate, as well as possible by a series of pictures, the positions and expressions in the various phases of his constant movement.

The symptoms produced by lesions of

A SERIES OF PHOTOGRAPHS ILLUSTRATING THE PROGRESSIVE PHASES OF MOVEMENT AND FACIAL EXPRESSION THROUGH WHICH THE PATIENT IS CONSTANTLY PASSING.



I.



II.



III.



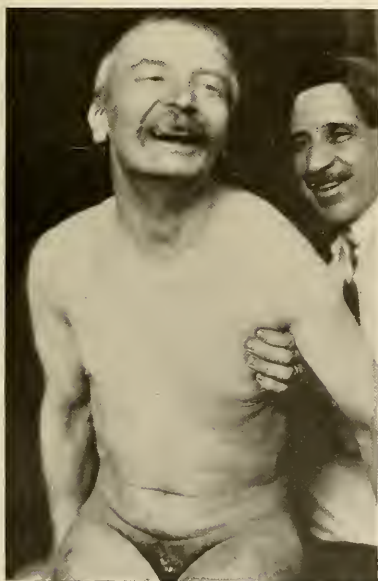
IV.



I.



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IV.

the optic thalamus were first adequately described by Déjerine, and later more fully elaborated by Rousse. Only a limited number of cases exhibiting the thalamic syndrome have been reported in the literature, and but few of them show clearly the signs which are supposed to be characteristic. The findings in the case about to be described are not entirely thalamic, but the predominating features are probably explained on this anatomic basis.

Patient: J. W. W., born in Scotland. Aged forty-three, married. Occupation: Electrical engineer.

HISTORY. Chief Complaint.—Trembling of the limbs and continuous sleeping for three and a half months.

Family History.—Mother living, but of feeble health, aged eighty. Father died at the age of fifty from cancer of the rectum. Four brothers and five sisters all living and well. No history of tuberculosis or other chronic diseases in the family.

Past History.—Patient has always been well. He is subject to headaches, never very severe. He has had no gastrointestinal disturbances. He denies venereal diseases by name and symptom. Last October he had influenza, but made a good recovery. He has always been active and of a rather nervous temperament. He does not remember any acute infectious diseases in childhood.

Personal History.—Patient is an electrician and inventor. He is married; his wife is living and well. He had two children; one died through instrumental labor. His wife had one abortion during an attack of pneumonia. He eats his meals regularly, but sleeps very irregularly, often sleeping but a few hours at night, as he is in the habit of working late over inventions. He denies the use of alcoholic stimulants. He smokes about six pipes of tobacco a day. He has never used any drugs.

Present Illness.—Three and a half months ago he began to be very nervous and irritable. At the same time he developed a tremor of his hands. His ears pained him

for a week; this was followed by severe headache lasting two or three days. He stopped work and his physician ordered him to go to bed. The first night he could not sleep. The next night he fell asleep and for three weeks he slept almost all the time. It was possible to arouse him enough to take food, but he would frequently fall asleep while taking it or while being bathed. For the first two weeks he talked continuously during his sleep. When aroused he seemed rational, but his talk during his sleep was entirely irrational. After the first week, when aroused, he was frequently irrational. During the weeks he was sleeping he had a constant fever, at times as high as 104° . He had no vomiting or convulsions. His neck was not stiff. He had no paralyses of any muscles. After a time he gradually grew better, got up and seemed fairly well. He was not nervous or irritable, but somewhat indifferent to what was going on about him.

Two weeks ago his left arm began to twitch, with no particular movement, but flexion of the forearm was more frequent than any other. A few days later he began to have irregular movements of his head. At this time his left leg seemed weak. He could lift it but dragged it in walking. Soon the left leg also began the same irregular involuntary movements as in the arm and head. About this time the left upper eyelid drooped and he was unable to lift it. During the last four or five days the right eyelid has become the same as the left. There have never been any convulsive movements in the right arm or leg, while the movements of the left side have steadily grown worse; that is, they are almost constantly present when he is awake, but are made worse by excitement, and at some times of the day they seem worse than at others. His speech was normal until three weeks ago when a slight "slur" was noted. During the last few days he feels that his speech is more involved, but it seems to him to be more as though he did not have the

strength to talk. At times during the last two weeks he has been irrational and seems to be steadily growing more stuporous. He has had no incontinence of urine or feces. His appetite seems good.

Two weeks before admission he had an eruption around the waist line which he calls "hives." The eruption appeared in streaks and was accompanied by itching. It disappeared in a few days.

GENERAL PHYSICAL EXAMINATION

General Appearance.—A middle-aged man, well developed and nourished, lying dorsally in bed with his eyes closed. He does not appear to be in pain. At frequent intervals there is a spasmodic movement of the left arm, head and left leg. The left forearm is flexed on the arm. The left hand is tightly closed. His speech is rather indistinct. His mouth appears drawn to the left when smiling.

Head is well shaped. There are no tender areas over the skull, mastoid or sinuses. The *neck* is not stiff. The *eyes* are both closed; the patient is unable to open the left eye but can raise the right lid about half way. The pupils are slightly dilated, clear and regular. They react very sluggishly to light. There is a marked nystagmus to the left. The fundi appear normal. The *nose* is negative to external examination; there is no discharge. The *ears* are negative; the ear drums appear normal. *Mouth:* The lips are not cyanotic. The tongue is heavily coated. The teeth in the upper jaw are false; in the lower jaw they are poor. There is a moderate pyorrhea. *Throat.* The tonsils are not enlarged or inflamed.

Thorax is well developed; expansion is equal on the two sides and symmetrical.

Skin.—The axillary region of the left side seems more wrinkled and lax than on the right.

Lungs.—Resonance is normal throughout; there is no alteration in breath or voice sounds. No râles are heard.

Heart.—The apex is not seen or definitely

felt. The heart outline is normal, the left limit of dullness being 10 cm. to the left of the mid-line in the fifth interspace. There are no murmurs. The action is regular. Rate 100. The pulse is of good quality. Blood pressure 114-75.

Abdomen.—There are no areas of tenderness. The liver dullness extends from the fourth interspace to the costal margin. The liver edge is just palpable. The whole abdomen is held rather rigidly, but there is no localized muscular spasm.

External Genitalia appear normal.

Superficial Lymph Nodes are not pathologically enlarged.

Extremities.—The left arm is held rather spastically flexed at the elbow. The left leg is slightly spastic. There is tenderness on deep pressure over the muscles of the left calf and the left forearm. There is some hyperesthesia over the left forearm.



FIG. 1. GENERAL POSITION USUALLY ASSUMED. HEAD THROWN BACK AND INCLINED TO THE LEFT. LEFT ARM FLEXED AND LEFT HAND CLOSED. PERMANENT DORSAL FLEXION OF THE TOES ON THE RIGHT SIDE.

LABORATORY EXAMINATIONS

4/16/19. <i>Blood Examination:</i>	
Red blood cells	6,360,000
Hemoglobin	70%
White blood cells	15,800
Polymorphonuclear leucocytes	86%
Large lymphocytes	6%
Small lymphocytes	6%
Transitionals	1%
Eosinophiles	1%
4/17/19. <i>Blood Wassermann:</i>	
Alcohol antigen	Negative
Cholesterol antigen	Negative
4/17/19. <i>Blood Culture:</i>	
No growth	
4/17/19. <i>Spinal Fluid:</i>	
Amount	15 c.c.
Appearance	Clear, colorless
Pressure	Normal
Globulin	Trace
Lymphocytes	260 (The intern who made this count does not believe it is correct)

Culture	Negative				
Wassermann	2 c.c.	1 c.c.	0.6 c.c.	0.2 c.c.	
Alcohol	Negative	Negative	Negative	Negative	
Cholesterol	Negative	Negative	Negative	Negative	
4/21/19. Blood Examination:					
White blood cells					13,200
Polymorphonuclear leucocytes					84%
Large lymphocytes					7%
Small lymphocytes					18%
Transitionals					1%
4/21/19. Spinal Fluid:					
Pressure	Apparently decreased				
Amount	10 c.c.				
Appearance	Slightly blood tinged				
Globulin	Present				
Cell count	23 lymphocytes				
Colloidal gold curve	1, 2, 2, 3, 4, 3, 2, 1, 0, 0				
4/22/19. Urine Examination:					
Specific gravity	1.029				
Reaction	Acid				
Color	Dark amber				
Turbidity	None				
Precipitate	Slight flocculent				
Albumin	None				
Glucose	None				
Microscopical examination	Many amorphous urates				
5/5/19. Blood Examination:					
White blood cells					14,600
Polymorphonuclear leucocytes					73%
Large lymphocytes					9%
Small lymphocytes					17%
Transitionals					1%

NEUROLOGICAL EXAMINATION 4/16/19

VOLUNTARY MOTOR SYSTEM. Attitudes.—The patient is lying dorsally in bed; his head is thrown back and inclined to the left. Both eyes are closed; the left one appears more tightly shut than the right. His mouth is drawn over to the left side. The head is not held still but is rotated from side to side as though he were trying to bore a hole in the pillow. His left arm is flexed at the elbow and the fist is firmly clenched. This arm is constantly moved about, but the flexed position is generally maintained. The left leg is drawn up; the thigh is flexed at the hip and the leg at the knee. The limb is also in almost constant motion, being moved about in all directions. The right side of the face is somewhat flattened; the right arm and leg are usually extended and are moved about somewhat, but are not in as continuous motion as are the extremities of the left side. It is possible for him to hold his limbs and head quiet for about thirty seconds if he makes a strong effort, but he

is unable to inhibit the movement for a longer period. He is able to open the right eye to about half the normal extent, but cannot open the left one at all. Both eyes are drawn over to the left and there is nystagmus to that side. The mouth is drawn over more to the left and he is constantly making grimacing expressions which vary from a pouting of his lips, a rolling of his lower lip, to a forced smile. At other times there is a period of uncontrollable laughter. Forceful attempts to passively open his left hand and to extend his left arm are painful.

Gait.—The patient is unable to stand or walk.

Coordination.—Finger-to-nose and heel-to-knee tests on the right side are well performed. On the left side the hand cannot be opened so he attempts to touch the end of his nose with his knuckle. This is accomplished by coarse, irregular, rotary movements which end after considerable difficulty in placing the knuckle accurately on the tip of the nose. The incoordination becomes less as the hand nears the nose. The heel-to-knee test is carried out with the same difficulty on the left side, but in the end is accomplished with fair precision. There is a marked retardation of successive movements of pronation and supination on the left side; but they are normal on the right.

Skilled Acts.—He is able to salute perfectly with the right hand and makes a fairly normal attempt to do so with the left arm, though the fist is closed and it is accompanied by jerky movements. Speech is slightly slurred.

<i>Reflexes:</i>	<i>Deep</i>	<i>Right</i>	<i>Left</i>
Jaw		1	1
Pectoral		1	1
Biceps		2	1
Triceps		2	1
Wrist		1	1
Radial		1	1
Ulnar		1	1
Patellar		3	1
Suprapatellar		3	1
Achilles		3	1

(1 = slight; 2 = active; 3 = with transient clonus)

(1 = slight; 2 = active; 3 = with transient clonus)

<i>Superficial</i>		
Ciliospinal	Absent	Absent
Supra-umbilical	Present	Present
Suprapubic	Present	Present
Upper lateral abdominal	Present	Present
Lower lateral abdominal	Present	Present
Plantar flexion	Absent	Present
Babinski	Present	Absent
Chaddock	Present	Absent
Oppenheim	Present	Absent
Gordon	Present	Absent
Schafer	Present	Absent

There is transient patellar and ankle clonus on the right side.

Abnormal Involuntary Movements.—These have been described under the head of general appearance and consist of a choreo-athetosis which is limited to the left side and most marked in the head, face and arm.

Muscle Strength.—This is rather difficult to test on account of the constant movement of the left side, but there seems to be no definite loss of strength on either side.

Muscle Status.—There is no apparent loss in volume or change in contour of any portion of the body (except for the flattening of the right side of the face).

Abnormal Associated Movements.—None are present.

NERVE STATUS.—Nothing abnormal noted.

GENERAL SENSORY.—Pain, temperature, vibratory, muscle-tendon sense and stereognostic perception are all normal. There is some hyperesthesia over the left forearm and hand and distinct tenderness on deep pressure over the muscles of the left forearm, left thigh and calf.

Subjective Sensibility.—He complains of severe burning pains in the left forearm and hand which are present most of the time. Passive extension of the fingers or forceful extension of the left wrist cause him to cry out with pain.

CRANIAL NERVES AND HEAD. *Olfactory Nerve and Nose.*—Normal.

OPTIC AND OCULOMOTOR APPARATUS.—*Visual acuity* cannot be accurately esti-

mated. *Perimetry*, roughly tested, seems normal. (The patient was seen by Dr. Tilney before his admission to the hospital and at that time had a left homonymous hemianopsia.) *Eye grounds:* Both optic disks are normal; there are no hemorrhages. The *pupils* are moderately contracted, regular, equal; reaction to light and on accommodation is limited. The left eye is entirely closed; the right one is often closed but may be opened about half way. There is no evident exophthalmos or enophthalmos. There is a deviation of both eyes to the left and a limitation in his ability to move them to the right. There is a moderate horizontal nystagmus with a slow movement to the left.

Acoustic Nerve and Ear.—The hearing is normal on both sides. Air conduction is greater than bone on each side.

Trigeminus Nerve and Mouth.—The jaw reflexes are normal. Both conjunctival reflexes are present. There is no evident weakness of any of the muscles of mastication.

Facial Nerves and Face.—Taste on the anterior two-thirds of the tongue is normal. There is a slight flattening of the lower portion of the face on the right side.

The Glossopharyngeus, Vagus Nerve, Pharynx and Larynx.—The uvula is in the median position. The palatal and pharyngeal reflexes are normal. The quality of the voice is normal. There is no difficulty in swallowing.

Spinal Accessory Nerve.—There is no evident weakness of either sternomastoid or trapezius muscle.

The Hypoglossus Nerve and Tongue.—The tongue is protruded in the mid-line, and there is no trophic disturbance, tremor or twitching.

Mentally he is at present surprisingly clear and bright, answering questions promptly and correctly and showing a keen sense of humor. At times he has delusions, believing that they are trying to starve him and not wanting him to have water or to see his wife. Occasionally at night he is quite delirious, talking loudly

and incoherently and attempting to get out of bed. He is perfectly oriented, but his memory for recent events is quite faulty. There is marked emotional instability resulting in uncontrollable laughter or crying. He shows interest in the condition of the patients in the beds next to his, and cried at the sight of one of these patients greeting his wife. He is surprisingly indifferent to his own condition and if questioned about it expresses the feeling that he will entirely recover. The constant movement and contortion of his head, arm and leg does not seem to tire or distress him very much.

SPECIAL EXAMINATIONS AND CLINICAL NOTES

4/18/19. *Eye Examination* (by Dr. Shoenberg):

1. The patient keeps both eyes closed. If told to open them he does so, but shows a slight ptosis on the right and a marked ptosis on the left.

2. Position of the eyes is that of conjugate deviation to the left of an irritative nature. Motility is good up, down and to the left; slightly insufficient to the right. There is slight insufficiency of convergence.

3. Pupils dilate to dark, but the reaction to light and on accommodation is very much reduced.

4. Corneal sensibility is normal.

5. Fundi normal.

6. Vision cannot be taken on account of the patient's general condition.

4/22/19. *Eye Examination* (by Dr. Shoenberg):

1. Vision is about 10/15 in the right eye; 10/30 in the left.

2. Patient observes color—red and green—in each eye.

3. Conjugate deviation to the left has disappeared but there is still slight difficulty in looking to the right.

4. There is marked insufficiency of convergence.

5. Fields of vision roughly taken are normal. No hemianopsia.

4/22/19. Since the patient has been in the hospital he has been irrational the greater part of the time. He has delusions, especially in regard to the medical attendants, believing that they are persecuting him and do not wish him to get well. Two days ago he would not remain in bed and had to be restrained. Yesterday he seemed more normal and easier to manage, but to-day he is quite obstinate and refuses all forms of medication. The symptoms have remained practically the same for the past few days, the temperature ranging from 100° to 101°. He has complained of no pain. He had to be catheterized once. The choreo-athetotic movements of the head, left arm and leg continue. These are more violent when he becomes excited, and disappear entirely when he is asleep. Occasionally when drowsy he shows signs of catatonia, holds his arms in the air and his mouth open and remaining in this position for several minutes.

He is now able to open both eyes to some extent, the right more than the left.

4/25/19. The movements of the left side continue without apparent change. Mentally he is much clearer and more reasonable. At times he has a good deal of pain in the left arm and leg. He is now able to open both eyes fully, though he very often keeps the left one entirely closed and the right one partly so.

5/5/19. The patient has been sitting up in a chair for about one-half hour at a time for the past few days. In general, he seems somewhat better. Often his movements are not as violent as before, but they are made worse by any excitement. He has a distinct antipathy to popular music and if it is played on the phonograph in the ward he becomes very excited. He enjoys classical music, and if it has any effect upon him it is a quieting one.

5/15/19. The patient has improved very much since the last note. He is almost constantly entirely rational. At night he

sleeps well, is quiet and has no delirium. The movements of the left side are much less violent and it is possible for him to inhibit them entirely by a strong effort for a period of about two minutes. He is able to extend the fingers of his left hand fully except for the little finger which remains partially flexed. Dysesthesia is present on the left side but is not as pronounced and troublesome as formerly. There is no ptosis on either side. The nystagmus has disappeared and the pupillary reactions are normal. He is able to read the larger print in the newspaper, but finds that the focal distance for small print is about double what it was before his illness commenced. The visual fields are not contracted. The weakness of the right side of his face is difficult to demonstrate and has practically disappeared. The patellar and Achilles reflexes on the right side remain active, and the Babinski and its modifications are still present, though his great toe is no longer constantly in the position of dorsal flexion. The left hemiataxia is still marked.

The treatment he has received has been entirely symptomatic, consisting of sedatives administered at irregular intervals as required. Chloral hydrate and paraldehyde have been more quieting than bromides or drugs of the veronal series.

SUMMARY

The salient points in the history are the gradual onset of an illness characterized by marked stupor, somnolence, delirium and fever, later by choreo-athetotic movements of the left arm, burning, pricking sensations in the left arm and leg, weakness of the left leg, ptosis of the left eye, involuntary movements of the head and left side of the face, ptosis of the right eye, involuntary movements of the left leg; then lessening of the ptosis in the right and then in the left eye, return of normal strength in the left leg, improvement of mental state, diminution in violence of choreo-athetotic movements.

The important findings on neurological examination were: choreo-athetotic movements of the left side, most marked in the head, face and arm, normal reflexes on the left, increased reflexes on the right, with a positive Babinski and ankle clonus; bilateral ptosis most marked on the left, slight weakness of the lower portion of the face on the right, conjugate deviation of the eyes to the left, nystagmus to the left, slight diminution in ocular movements to the right, pupils which react sluggishly to light and on accommodation, probably a left homonymous hemianopsia, ataxia of the left arm and leg.

In consideration of the history of prolonged somnolence, lethargy and transient cranial nerve palsies, together with the spinal fluid findings, the clinical condition seems to be quite clearly epidemic encephalitis. The neurological findings cannot be explained on the basis of a single lesion, but the predominating signs seem to conform closely enough to the thalamic syndrome to allow of a diagnosis of a lesion of the right optic thalamus.

The accepted signs of a thalamic lesion are unilateral: Pain, anesthesia (deep, and frequently superficial), ataxia, astereognosis, choreo-athetoid movements. Other findings from involvement of neighboring structures include hemiplegia, hemianopsia, disturbances of ocular movements and of the pupillary reflexes.

In this patient dysesthesia and pain were present. Hemianesthesia was not present, at least during his stay in the hospital, there being a moderate hyperesthesia to touch over the left forearm and hand and pain on pressure over the left forearm and calf. There was no astereognosis, the patient recognizing the size, shape, consistence and surface characteristics of objects placed in either hand. He was also able to reproduce with his right hand any position in which his left hand and fingers were placed. Ataxia and choreo-athetoid movements were present. There were disturbances of the ocular movements and pupillary reactions, and before he

entered the hospital weakness of the left leg and a left homonymous hemianopsia. The increased deep reflexes and Babinski on the right side must of course be due to a separate lesion in the corresponding pyramidal tract.

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THE PSYCHONEUROSES AND INTERNAL SECRETIONS*

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INTRODUCTION

Perhaps the bleakest chapter in the whole domain of medicine is that on the treatment of nervous diseases. Great progress has been made in pathology and diagnosis but, with the possible exception of syphilis of the nervous system, none in therapy. We often pride ourselves on the brilliancy of diagnosis and accuracy of localization but lose sight of the fact that our patients are little interested in either. We stand helpless before every attempt at cure and watch every case go through its tragic cycle without being able to interrupt any of the turns. Our blind efforts often appear as comic sides to serious situations and would be truly humorous if they were not so tragic.

The psychoneuroses, while not so inexorable or relentless, form no exception to the general run of neurological cases. We see patients coming to the office or the clinic for months and years, merely showing ebbs and flows of symptoms but no real cures or even encouraging progress. We use bromides as sopors to our conscience or hypodermic injections as props to our incomes or high frequency as spectacular suggestion to patients; but, if we are honest with ourselves, we recognize the blindness of the procedure and the futility of our efforts. That some patients do get well cannot be held in favor of our treatment: we have not cured. We know neither the reason for their becoming sick nor the why of their getting well. It is true that in recent years psychoanalysis has attempted to supply an answer to both. Nor is it the purpose of this essay to quarrel with that method of diagnosis and therapy. As a matter of fact both normal and abnormal psychology are greatly indebted for

progress to the psychoanalytic method if not to some of its exponents. But that too has failed to supply the mainspring behind the assumed conflict of emotions. Why does one and the same experience arouse a conflict or dissociation or defense mechanism in one and not in the other? We don't know. Put the blame on heredity, I suppose. The same questions may be asked as to general immunity. The beautiful side-chain theory of Ehrlich if closely analyzed is seen merely to beg the whole question. Why does one man develop amboceptors and complements and antibodies in response to microbic invasion and not another? To speak of overwhelming invasion or great virulence or excessive shock is merely to use apologetic expressions for our inability to understand a situation. They half explain, but do not go to the root of the matter.

In stating the problem in the form of questions it is not my intention to discourage, or exhibit too great a fatalistic pessimism. Quite the contrary. I believe we are on the verge of epoch-making changes in the domain of medicine as in other domains. The trouble, it seems to me, is that we have looked for causes too much outside of and not enough within the human organism. From now on we shall pay more attention to the inner processes without for a moment ignoring the tremendous importance of environmental forces. I refer of course to the rôle of the internal secretions. Before, however, trying to correlate that rôle with the subject of psychoneuroses it may not be out of place briefly to epitomize what we actually do know of the internal secretory processes and then see how far those facts lend themselves in explanation of our theme.

There is a tendency at this day to explain

* Read at the Clinical Conference, May 22, 1919.

everything we don't know by internal secretions. There is great danger in such a tendency. It may or may not be right, but we have nowhere near enough facts to prove that it is. Somehow the human mind has the great failing of running to extremes. In the case of the psychoneuroses the psychoanalyst will have it that he has a patent on truth, and now the internal secretion student claims to have obtained a copyright on the same elusive subject. The probability is that each has a very important function to perform, and while the psychoanalyst approaches the problem after it has become one, the internal secretion student seeks to attack it before its incipency. I am of the opinion that we will have reached more solid ground if we can establish an organic basis behind some of the psychoneuroses and perhaps seek in the internal secretions an explanation of their genesis.

Before reviewing briefly the knowledge we have of the effects of the internal secretions it is well to point out that too much emphasis must not be laid on facts. Many of those so-called facts have been proved on repeated experimentation to be no facts at all, and no doubt a number of those unassailable facts which we swear by at present we may swear at in the future. One need merely point to the recent experiments which tend to show that adrenalin, far from being the great high blood pressure producer we so confidently believed it to be, is in reality a lowerer of blood pressure, at least in the small quantities in which it circulates in the blood. But while we have not enough facts and must be warned against undue speculation a little theorizing may not be out of place.

THE INTERNAL SECRETIONS

There are in the body several glands which pour out their metabolic products into the blood stream and seemingly set up changes which directly or indirectly affect the whole organism. Many organs

secrete certain chemical substances, called hormones, which in some way affect the activity of other organs or possibly indirectly also the body in general. We have known for a long time that the nervous system, both vegetative and cerebrospinal, controls the workings of the body. The ancients believed that the bodily organs controlled the workings of the nervous system. We laughed at them for a long time. Galen is no doubt laughing in his tomb at our modern reversion to his ancient conception.

We know that there is a reciprocal relation between the various glands of internal secretions. The thyroid, for instance, inhibits the activity of the pancreas which in turn inhibits the activity of the chromaffin system, that is, the medulla of the adrenals. The thyroid and posterior lobe of the pituitary also activate the chromaffin system. Thus excessive secretion of one gland may disturb the harmony normally existing between all the glands and both directly and indirectly lead to the manifestation of abnormal signs and symptoms. Therefore, although the thyroid does not control sugar metabolism, we may get a glycosuria in hyperthyroidism from the fact that the activity of the pancreas has been inhibited and that of the adrenal doubly increased because the pancreas fails to inhibit and the thyroid activates it. Besides excessive function, however, there is such a thing as perverted function or dysfunction.

As to metabolism, it is generally accepted that the pancreas governs carbohydrate assimilation and the adrenals preside over glycogen combustion. The thyroid aids in albumin metabolism, probably increases gaseous exchange, influences heat regulation in some way and possibly presides over resistance to infections. Both the chromaffin system and hypophysis may also have something to do with heat regulation. The glands of internal secretions no doubt influence the growth of the body. For instance, early loss of sex glands leads to tallness, diminished function of the

thyroid at an early age leads to dwarfism and poor development, while early absence of the hypophysis brings about stunting, hypoplasia of the genitalia (the interstitial glands) and mild eunuchoidism. The pineal seems to govern bodily growth, as disturbance of it in children leads to macrogenitosomia. Hyperfunction, on the other hand, of the sex glands and the suprarenal cortex early in life gives excessive growth and increased genital function. That the glands of internal secretion control the secondary sex characters, such as hairiness of the body, fat distribution, etc., is of course well known. It should be added, however, that there may be slight disturbances of function which do not lead to visible changes yet undoubtedly disrupt the general bodily harmony and possibly explain the mysterious onsets of psychic or functional conditions.

There is a strong bond of union between the glands of internal secretion and the nervous system. It is difficult to say which is the cart and which is the horse. Does the nervous system stand under the control of the glands or are the glands dominated by the nervous system? One sees psychic irritability, irascibility and even manicuephoric states in Basedow's; apathy in myxedema, mental weakness in Addison's and mental strength in sexual potency. The vegetative nerves are also influenced by the products of internal secretion. The product of the medulla of the adrenals stimulates the sympathetic system while cholin, which is assumed to be the product of the adrenal cortex, possibly influences the autonomic or extended vagus system. We know that thyroïdin heightens the vagal tone and leads to irregular breathing, fast pulse, low blood pressure, sweating and various ocular signs. The posterior lobe of the pituitary irritates the pelvic nerve, causes frequent urination, renders the uterus excitable and increases the blood pressure, while pituitrinum glandulare lowers the pressure. Excessive thyroid gives spasm of the stomach, intestines,

bladder, heart and ciliary muscles. Increased sex activity brings about increased vegetative function and its influence may be seen in so-called vasomotor ataxia and lability of the vegetative system in the climacterium.

Some glands apparently are under direct control of the nervous system while others exhibit more or less independence. Both the adrenals and the sympathetic belong to the chromaffin system. Thyroid transplants are unsuccessful apparently because of our inability to subordinate them to nerve control. Sex glands, on the other hand, seem to be very independent of nerve control as heterotransplants without new nerve connections function very well. However, there possibly is a sex center in the subthalamic region. But that the glands are under nervous control can be seen from the influence of emotions such as fright and worry on diseases like Basedow's and diabetes. It must be remembered also that there may be functional increase without actual disease. In this connection the work of Crile and Cannon may be mentioned as showing the reciprocal relation between the glands and the nervous system. And here comes in the question whether the emotions are the result or the cause of internal secretory changes. If violent emotion causes an increased output of adrenalin, can an increased output of adrenalin cause a violent emotion? The James-Lange theory assumes that the emotions are the result of bodily changes and not the cause.

One observes reduced excitability of the vegetative nerves in melancholia and increased activity in mania. In many psychoses we find altered metabolism. Some vasomotor neuroses show increase in temperature, and in Raynaud's disease one may get polyuria beside a rise in temperature. Not uncommonly one observes transitory edema, urticaria and erythemas in vegetative neuroses. In a "debile" nervous system such as asthenia we observe disturbances of gastric secre-

tion, constipation and enteroptosis. Grave depressions are said to limit thyroid and adrenal secretions.

In reviewing the above facts, which are well known to everybody, the object has been to point out the close relation between the glands of internal secretions and the physical and mental functioning of the organism as a whole, and the further bearing they may have on the psychoneuroses. Some investigators have even posited the existence of individuals as ductless glandular "debiles, stables, and labiles." By "debiles" they mean individuals who show tendencies to infectious diseases and slight resistance to internal and external physical and mental stress. The "stable" of course is one who shows well balanced responses and ability of proper adjustment. The "labile" ductless glandular person exhibits variations in bodily weight, fluctuation in emotions with transitory glycosuria, altered psychic states; women with signs of acromegaly or Basedow in pregnancy and menstrual disturbances such as dysmenorrhea, amenorrhea or menorrhagia.

The many signs and symptoms which one sees in the psychoneuroses closely resemble those observed in vagotonia and sympatheticotonia. But the indefinite and ever changing functional diseases even more closely bespeak disturbances of internal glandular activity, at least in a great number of instances. One does not have to note actual disease syndromes, but merely beginning signs of disturbed functions. Such signs are not infrequently present in the psychoneuroses and I shall refer to them in quoting cases and analyzing their probable etiology.

It may be possible to appease the wrath of the psychopathologist or perhaps satisfy his unconscious desire by giving a physical basis to his analytical speculations. Who knows but what the emotional sexual conflicts really have an organic background and that in the disturbance of internal glandular secretions? Perhaps a disturbance in childhood of the pituitary

function which indirectly gives rise to disturbed sex function may be the cause of the formation of an infantile complex. It has been shown that feeding pituitary extract to preadolescent animals leads to early ovulation and spermatogenesis. "These observations, coupled with the knowledge that the secretory discharge from the hypophysis may be elicited through the sympathetic nervous system, suggest that the liberation of a chemical messenger may account for the recognized effect of the emotions upon the sexual sphere." (Cushing.) As both the pineal and the pituitary influence sex, disturbances in their structures in early childhood, not sufficient to exhibit actual physical signs, may be the starting points of altered or labile mentality and the precursors of early mental conflicts. The ultimate development may be affected by the altered sexual activity. And to quote Cushing once more (*Amer. Jour. Insanity*, 1912, No. 5):

"It is quite probable that the psychopathology of everyday life hinges largely upon the effect of ductless gland discharges upon the nervous system. This is particularly worthy of consideration in the study of child psychology in its relation to puberty and adolescence, especially in those individuals in whom there is some underlying, possibly inherited, functional deviation of the chemistry of the internal secretions. . . . If, therefore, we are to swallow the Freudian doctrines whole—a difficult morsel for many—and are to interpret hysteria and the psychoneuroses solely as the resultant of early mental conflicts and compromises between libido and its repressions, it will easily be seen that any secretory duration which on the one hand excites, or on the other diminishes, sexual activities, must be an important element in modifying the terms affecting the ultimate compromise. . . . It is hoped that some serological test, possibly in the direction of Abderhalden's investigations on the serodiagnosis of pregnancy, will serve to give us a chemical

reaction of diagnostic value, at least, for states of overactivity of individual glands . . . and we possibly may come to attach importance to the findings of pathological serology far greater than that which in the past we have given to cytological changes shown in the microscope." The serological tests may overcome the present difficulty of quantitatively determining the blood content of internal glandular secretions and help us to determine also in the psychoneuroses the existing imbalance, and possibly serve as an explanation of symptoms.

With these general considerations in mind let us examine the symptoms caused by disturbance in individual glands and see what reasons we have for presupposing such disturbances in the psychoneuroses. If in a given case which hitherto has been labeled purely functional we can discover some signs of altered endocrine function it may not be overbold to assume such an alteration as a basis for the symptoms. And further the disturbed function may not be alone or primarily in the gland to which the observed signs and symptoms are referable. We may be dealing with a pluriglandular condition, or disturbance in one gland as the direct or indirect result of disturbance in another. Should the observation prove to be correct we may be encouraged to institute rational therapy directed toward the correction of the functional disturbances. I have collected blood pressure readings in about one hundred and fifty psychoneurotics of all sorts, with a view of finding an index for classification and treatment, but thus far the results are not conclusive and the number of cases is not sufficiently large to base any definite opinion on it.

CASE HISTORIES

The following cases are cited as illustrations of psychoneurotics who gave signs and symptoms referable to the pituitary gland. I shall leave out irrelevant details, giving only abridged histories.

CASE I. A. N., female, age thirty-four. Complaint: Irritability, tiredness as the day progresses, spells of weakness, trembling, forgetfulness, laughing and crying easily, pressure in frontal region of the head, general aches and pains all over the body, particularly the limbs, and excessive worry. Menses normal. Neurological examination negative except for a fine rhythmic tremor of the fingers, diminished corneal sensation, slight temporal contraction of the fields of vision, $L > R$, margins of disk not clear. Marked hairiness of upper lip and numerous coarse hairs on face; arms markedly hairy, male pubic. The skin is flabby, excessive panniculus and large breasts. Dermographia. Blood pressure 175 S. 110 D. Accentuated second aortic. Diagnosis: Psychoneurosis, dyspituitarism and hypertension. She was put on whole gland pituitary extract and thyroid. An x-ray, urine examination and Wassermann were ordered. The x-ray report: "Sella has normal diameters. There is, however, a tendency to approximation of the anterior and posterior clinoid processes, which undoubtedly would retard the development of the hypophysis. The soft tissues of the face are thickened and there is an upper jaw prognathism." Wassermann negative, urine showed trace of albumin, no casts or sugar. Two weeks after the first visit the patient returned much improved, the blood pressure was reduced to 160-100. Continued use of pituitary and thyroid has kept up the improvement to this day.

CASE II. R. P., female, age twenty-one, single. Complaint: Depressed, sad, cries, has no courage, is not interested in anything, has heaviness on top of the head. Pains all over. The history dates back nine years to the age of twelve. Then she saw her parents in the act of intercourse and she became depressed, dreamy, etc. Soon she learned to masturbate. Had several periods like the present one. Thinks she'll not get well. Is afraid she'll go crazy and is sure masturbation will be her undoing. Has excessive libido and

constantly fancies herself in actual intercourse. Her menses are irregular, three to four weeks, scanty of late and accompanied by pain on top of the head. Evidently a gold mine for the psychoanalyst. Neurological negative except for contracted (?) fields of vision, acuity 20/70 O. D. 20/100 O. S., disk not clear, probably normal. Male pubic hair, marked facial hirsuties and hairiness of arms. Dermographia. Blood pressure 130 S., 70 D. X-ray shows the clinoids almost impinging upon each other. There is hardly any doubt that there is a disturbed function of the pituitary, and who knows but what the psychic symptoms are intimately connected with it. This patient has been left to the tender mercies of the psychoanalyst. (Since writing this paper I have been informed that she has greatly improved with the kind treatment of Dr. Bernard Glueck.)

CASE III. G. G., boy, age thirteen. Complaint: Utterly unmanageable, fits of violent temper, frontal headache. Began suddenly six months before. On being refused an expressed wish for something he throws himself to the floor, screams, kicks, uses abusive language, then holds his breath till he becomes blue in the face, keeps on repeating certain words or phrases. He has had these fits repeatedly; is spoiled and petted. Weighs 112 pounds—a gain of 22 pounds in six months. Neurological negative except for absence of hair, even fine ones, on the body, female fat distribution over breasts, abdomen, hips, mons and thighs. Very small penis. Blood pressure 110-75. Owing to the incorrigible attitude of the boy no x-ray or eye examination could be made. The diagnosis was hypopituitarism plus hysteria (?). My report to Dr. Granet, who sent me the patient, was that the boy was more of a social than a medical problem and that the mother should be prevented from exerting a baneful educational effect on him. The strong hand of the truant officer or that of the juvenile court was in my

opinion more likely to bring about a cure. However, I suggested that pituitary extract might be tried, in view of the hypopituitarism. Dr. Granet adopted the more gentle method of treatment and a few weeks on pituitary extract caused a marked change in the boy. His character altered to such an extent that he has once more become the docile young fellow that he was.

Parenthetically it should be added that in most of our treatments we are exceedingly prone to invoke the *post hoc ergo propter hoc* proposition. It is very difficult to dissociate the personal influence of the physician from the pharmacological effect of the drug. This is particularly true of psychoneurotics who are so amenable to suggestion. It may readily be argued that any drug or any treatment may give favorable results, depending upon who employs it. While that is true enough in general, and the fact is apologetically made use of by the quack, there seems to be no doubt that the favorable result attained in Case III was directly due to the specific effect of the extract on a deficient pituitary. Certainly the influence of the writer was nil, as the boy bolted from the office the minute the examination was over.

CASE IV. M. D., male, age twenty-six. Complaint: Pain all over the body. Headache, feeling of hot and cold, loss of appetite, constipation, occasional vomiting, tiredness, worry and excessive sweating. Six months before suddenly had an attack of great weakness with pains in the legs, and was taken to a hospital where he remained for ten days. Since then the above symptoms have come on. Has worries over his people in the Russian war zone and is afraid of insanity. He can't be in company and wants to be alone. He is easily frightened and his memory is poor. Masturbates occasionally; never had intercourse. Neurological negative. Has large breasts and effeminate body and small penis. Blood pressure 120-80. Diagnosis: Psychoneurosis with precox makeup and hypopituitarism. Treatment con-

sisted of pituitary extract. He felt well while taking it, but the symptoms returned on ceasing to take it. Have lost track of the patient—not a very great compliment to my therapeutic endeavors.

One or more of the symptoms of hyper- or hypopituitarism may accompany a psychoneurosis and possibly account for it. One need not wait either for an acromegaly or dystrophia adiposita genitalia to make a diagnosis of pituitary disease. Some of the following early signs and symptoms may be looked for. In hyperpituitarism there is an accentuation of function of the thyroid, such as sweats and tremors, increased activity of the interstitial sex glands as shown in the secondary sex characteristics; loss of genital function, that is, impotence or absent libido; amenorrhea, etc., after preliminary increase; bushiness of eyebrows, hairiness of body. Many of these symptoms may be secondary to disturbance in sex glands, such as mild acromegaly seen in pregnancy. Early somatic signs are rheumatoid pains, night pains, acroparesthesia, sensation of heat in the fingers, swelling of joints and frequent urination.

In hypopituitarism the symptoms which ultimately may lead to a typical dystrophia are inhibition of the activity of vegetative nerves, polyuria and polydipsia. The fat accumulation usually is in the supra- and intraclavicular regions, "cuffs" around the malleoli, collar of fat around the neck, adiposity in the abdomen and mons, large breasts which are poor in gland structure. The skin is delicate. The penis, testes, prostate, clitoris and labia are small. Lack of hairiness of the body; absent libido, impotence and cessation of menstruation. In most of the cases of early involvement of the pituitary even when the physical signs and symptoms are not marked an x-ray of the sella may show organic changes.

It will be noted that many of the symptoms enumerated constitute a common picture of so-called pure psychoneurosis. But more frequent than those are

the functional conditions with thyroid backgrounds. The following cases will illustrate the point.

CASE V. R. C., female, married, age thirty, complained that for eight weeks she has felt miserable, she cries a great deal, is tortured by the thought that she may be responsible for the death of her brother, who is ruptured, because she induced her husband to employ her brother, and that lifting heavy weights will aggravate the rupture and kill the fellow. She thinks that she has sinned against her mother by inducing her brother to run such dire risks, yet she does not like her mother. The onset of the illness was sudden. Since then her libido has disappeared, she sweats a great deal and tires easily. Obviously we deal with an obsessional neurosis, but physically the patient shows marked rapid tremor of the fingers, strong dermatographia, enlarged thyroid, a pulse of 120. She is quite sure that her neck was not large before the onset of the present condition. To the tentative diagnosis based on the history we must add at least hyperthyroidism if not a true Basedow.

CASE VI. J. B., female, married, age twenty-five, complained of choking sensation in the throat, dryness and heaviness in the chest, shivering and shortness of breath. The onset was sudden, following the news that a friend of hers became insane. At first she feared insanity and now is afraid of dying. The menses last eight days and are very profuse. Physical examination was negative, as usual in anxiety neuroses, but there was a slight tremor of the fingers, diminished corneal sensation, marked dermatographia, enlarged right lobe of the thyroid, pulse 120, blood pressure 130-65. I ignored for the moment the anxiety state and treated the hyperthyroidism by putting the patient to bed for ten days and giving her tr. belladonna and chloral. She improved a good deal and I think is now entirely well.

CASE VII. S. T., female, married, age twenty-eight, complained of a sense of confusion, vague pains all over the body, occasional dizziness and vomiting. The condition came on suddenly after confinement which was followed by an attack of influenza. She has been very much "afraid" ever since. Menses are scanty, libido weak. She has occasional diffuse headaches. Neurologically negative, but she has a tremor of the fingers, a Stellwag, marked dermatographia, excessive perspiration, enlarged thyroid and pulse of 100. Blood pressure 130-84. There is no doubt that in this case too we are dealing with a hyperthyroid beside the psycho- or anxiety neurosis.

There are many symptoms of hyperthyroidism which without combining to give a Basedow may stimulate or perhaps initiate a neurosis. The following are not uncommon: Palpitation, lump in the throat, tremor increased by emotions, weakness, pains all over the body and extremities, headache, irritability, emotional changes and excitement. Loss of weight, increased sweating, feeling of heat, falling out of hair, increased salivation, hypo- or hyperacidity and diarrhea. The ocular signs, of course, are important.

Disturbance of function of the thyroid may show itself in diminished activity and one may hear patients complain of vague pains, headache, lassitude, sensation of cold, constipation, falling out of hair and menstrual disturbances. One not uncommonly sees a combination of these symptoms in patients whom we conveniently label neurotic. Some time ago I reported a case in which, following pregnancy, there developed headache, tiredness, dryness of the hands, menorrhagia and falling out of hair. A diagnosis of hypothyroidism followed by thyroid medication served to clear up the condition. It may be added that hypothyroid conditions may follow exhaustion such as is entailed in pregnancy and parturition, and the hyperthyroid state of pregnancy is at times followed by diminished function.

There are some cases in which one sees combined hypo- and hyperthyroidism; perhaps dysthyroidism is a better term for them.

In connection with the thyroid one might mention the parathyroids, in impaired function of which one finds abnormally increased irritability of the nervous system. In mild cases may be observed hyperexcitability of the motor, sensory and vegetative nerves and occasionally even paresthesiæ. At times one gets a Chvostek in cases which do not at all show the tetanic picture one expects. Hypoparathyroidism may be the cause of a neurosis in pregnant women or during the puerperium and lactation.

The suprarenal glands no doubt are frequently the cause of what, for want of clear recognition, we designate as psychoneurosis. Certainly many of the older neurasthenias bear a very close resemblance to hypoadrenemia. If one takes the early history of a typical Addison one finds easy fatigability, apathy, headache, poor sleep, depression, nausea, constipation or diarrhea and loss of weight. This array of symptoms is so commonly seen in "psychoneurotics" that one is frequently tempted to make a diagnosis of early Addison. Indeed, there is no reason why a disturbed adrenal may not stop with the production of just these symptoms and not go on to bronzing of the skin and mucous membranes, asthenia, etc. And bearing in mind that infectious diseases, particularly diphtheria, do attack the adrenals there is every reason to believe that we often meet with unrecognized cases of hypoadrenemia. The Sergent line which is supposed to be pathognomonic of this condition, although useful, occurs so frequently that one should place only limited reliance on it. So too the blood pressure is not always diagnostically indicative of hypoadrenemia. In trying to correlate blood pressure findings with anxiety neuroses, obsessional condition, depressions and general psychoneuroses, I could not make sure that there was great

significance. Recent views, moreover, tend to show that the adrenalin of the blood does not raise blood pressure but actually lowers it. A few cases may illustrate possible adrenal involvement, although it should be added that this gland is often secondarily affected by the pituitary and sex glands.

CASE VIII. M. K., male, age thirty-eight, complained of impotence and general weakness, loss of weight and poor sleep. He is very much depressed. The impotence came on following the death of his wife. Physical examination was negative except for hairiness of the whole body, profuse sweating, white adrenal line and a blood pressure of 104 S. 70 D., which, considering his age, is very low. The diagnosis was psychic impotence plus hypoadrenemia.

CASE IX. E. R., male, age thirty-one. His complaint dates back many years. It began with backache and headache, then followed by pains all over the body and shortness of breath. At times vomiting. Tires easily, has spots before the eyes on doing continuous work. Any physical or mental exertion makes him "all in." On several occasions he suddenly felt weak and dizzy, and although he reeled he did not fall. Once he was assaulted and sodomy was practised on him. Lost a great deal of weight. He is impotent. Physical examination was negative except for marked weakness, white adrenal line and a low blood pressure—98 S. and 80 D. A markedly relaxed anal sphincter bespeaks more than one assault of sodomy. The diagnosis was sexual neurosis, if not perversion, and hypoadrenemia.

These two cases implicate, perhaps not very conclusively, the adrenals, that is the medulla or chromaffin tissue. But there are cases in which the suprarenal cortex is involved. In case of hyperfunction the result is increased hairiness of the body and accentuated secondary sex character, while hypofunction leads to a diminution of those characters. Hyperfunction of the medulla

may lead to arteriosclerosis, atheromatosis and glycosuria.

There is no doubt, of course, that the sex glands play an even more important rôle in the genesis of psychoneuroses than those illustratively discussed. The frequency of neuroses at puberty, during menstruation, pregnancy, lactation and particularly the menopause speaks strongly in favor of disturbed sex function. Unfortunately we have no clear-cut picture of disturbed function of the genital or interstitial glands, that is, not enough to guide us in making incriminating diagnoses. Perhaps a more detailed study of the menstrual periods would serve to elicit anomalies which ordinarily elude our observation. Ovarian adiposis, too, has something in it which is peculiar to itself. It should also be borne in mind that a disease like mumps frequently damages either the testes or ovaries, at times even beyond repair. However, thus far we must content ourselves with the general statement that the disturbed physiological balance is due either to secondary involvement of other glands of internal secretion or else to direct disturbance of the vegetative nervous system. There is sufficient reason to believe that in the latter, too, the internal secretions are at fault.

With reference to these nerves, recent investigators have tried to make them responsible for some of the psychoneuroses, and have described conditions in which the extended vagus, that is the autonomic system, is either hypo- or more commonly, hyperirritable and others in which the sympathetic proper is affected. To the first group belong the cases of vagotonia with slow pulse, respiratory arrhythmia, or even asthma, hiccough, cardio- or pylorospasm, constipation with unaccountable diarrheas, irritable pelvic nerve, von Graefe and diminished corneal or pharyngeal sensation, dermatographia, sweating, eosinophilia, etc. Cases of sympathicotonia particularly with vaso-motor changes are also described. There is no doubt that many psychoneurotics show a number of

the signs enumerated and many can be classified as vagotonics. Indeed, some respond very well to atropin giving pharmacological confirmation to the diagnosis, and I frequently make it a practice to prescribe the drug together with the panacea of the neurologist, those wonderful bromides. One case may illustrate the point.

CASE X. B. C., female, age twenty-seven. Her main complaints at present are dizziness, headache, constipation and chills all over the body. Close investigation shows that her dizziness is not really vertigo. She used to vomit considerably, but does not at present. An operation showed the abdominal organs to be normal. Always feels tired. Is afraid of becoming insane. Dreams a great deal of flying and running and going downstairs and falling from great heights. She urinates frequently by day; her menses are regular but last eight days and are profuse. On physical examination the only signs that were found were a von Graefe, diminished corneal and pharyngeal sensation, very marked dermatographia, slow pulse, that is 65 per minute, and low blood pressure, 95 S. 70 D. The diagnosis was psychoneurosis on a vagotonic basis. Bromides and atropin seemed to improve her a good deal. Unfortunately I could not follow up the case.

CONCLUSION

Without laying the slightest claim to originality I have tried to bring together well-known facts and to correlate them with other facts equally well known. Thus far the treatment of psychoneuroses has been so unsatisfactory, so blind and, if I may add, so irrational that one may be pardoned for seeking a little more solid ground to stand on. In clinging to the glands of internal secretions and seeking in them a source of rational therapy one need not ignore the great importance of environmental influences, of social maladjustments, of psychic and emotional conflicts, etc. In many cases one can find no other explanation than that given by the psychopathologist and there is no doubt that in selected cases his treatment is successful. But somehow one likes to feel that there is a physical-pathological basis behind some of the psychopathological states, and it may not be too presumptive to seek that foundation in disturbances of the glands of internal secretions. Certainly many cases illustrate the relation to a remarkable degree. Perhaps more detailed attention will serve to increase the number of such cases and it may be hoped that greater knowledge in the future will definitely place a number of the psychoneuroses in the realm of internal secretory disorders.

GLIOSIS OF THE BULB AND UPPER PORTION OF THE SPINAL CORD

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This case is reported chiefly because it illustrates the difficulty in the early diagnosis of a case involving muscular atrophy in the hands. The patient first came to the clinic about two years ago presenting simply muscular atrophy of the nuclear type of the muscles of both forearms and hands. Sensation was said to be normal at this time, and the diagnosis was pro-

posed and a further study. It was not until then that the dissociated anesthesia characteristic of syringomyelia was discovered and this diagnosis made.

Patient: J. M., born in Russia. Aged twenty-nine, single. Occupation, wood polisher in piano factory.

HISTORY. *Chief Complaint.*—Weakness



FIG. 1. FLATTENING OF LEFT SIDE OF FACE DUE TO MUSCULAR WASTING. DIMINUTION OF PALPEBRAL FISSURE AND ENOPHTHALMOS ON THE SAME SIDE.



FIG. 2. ATROPHY OF RIGHT HALF OF TONGUE.

gressive spinal muscular atrophy. About eight months ago an atrophy of the tongue was noted and also a bilateral Babinski, and then the diagnosis was considered to be amyotrophic lateral sclerosis. Two months ago a slight atrophy of the left half of the face was noted. At the same time the presence of the signs of paralysis of the cervical sympathetic on the left side was first observed, and proved conclusively that the former diagnoses were erroneous and that the case required

in the hands and arms with muscular wasting for the past six years.

Family History.—Father, mother, four brothers and one sister alive and well. No one in the family ever had a disorder similar to that of the patient. There is no history of tuberculosis, cancer, or any other chronic nervous or mental disease.

Past History.—The patient has always been well until the time of onset of the present illness. He does not remember having had any diseases of childhood or

any other acute infectious diseases. He has had no serious injuries, accidents or operations. He has never had headaches or any disorders of the respiratory, cardiac, gastrointestinal, or genito-urinary systems. Gonorrhea and syphilis are denied.

Personal History.—He is single and works in a piano factory, matching different colors of wood and polishing the wood. He used to get considerable out-door exercise, but at present gets very little. His habits of eating and sleeping are regular. He used alcoholic stimulants moderately until six years ago, but has



FIG. 3. POSITION ON ATTEMPTING TO OPPOSE THUMB AND LITTLE FINGER.

A normal hand on the left.

drunk nothing since. He was also a moderate user of tobacco before the onset of the present illness, but since then has not used it at all. He drinks one or two cups of tea or coffee a day, but has never used them to excess. He has never suffered from insomnia. His bowels move regularly once a day. Urination three to four times daily; no nocturia.

Present Illness.—Six years ago the patient noted that he had difficulty in flexing the little finger of the left hand. It felt weak. Gradually the other fingers of this hand grew weak and he could not grasp things readily. The weakness was espe-

cially marked in the fingers. He noted a gradual wasting of the fleshy portion of the hand progressing parallel to the weakness. Soon the left forearm grew weak and smaller. Six months later he noticed a dull pain in the left shoulder over the scapula area. It was continuous and did not radiate. This has continued without relief or change until the present.

About four years ago the right hand became involved in the same manner as the left, starting with a weakness of the little finger and progressing gradually until the whole hand and then the forearm were involved. These symptoms have progressed to the present time so that now he cannot pick up things with his fingers. He cannot carry anything in his left hand because he is unable to close it, and can carry only very light articles in the right hand, which he is still able to close. He is able to feed himself but has great difficulty in writing because he cannot hold a pen firmly. He has had no pain except that mentioned above, which is present in the back portion of his right shoulder. He has never had any difficulty in walking, and has noted no change in the size or contour of his limbs or any other portion of his body. He has no difficulty in swallowing or in speech. He believes he has lost 25 pounds in weight during the past six years. His present weight is about 170 pounds.

About six months ago he noted a wrinkling and shriveling of the right portion of his tongue. There have been frequent slight hemorrhages from this portion of the tongue. He also believes that the left half of his face is becoming smaller. During the past two years he has become slightly round-shouldered. At times he has noted small, black and blue swellings over his wasted forearms. They come on rather gradually and disappear after an interval of two or three weeks. They are never painful and are not the result of injury of any kind.

He has never had any difficulty with the control of his bladder or rectal functions.

GENERAL PHYSICAL EXAMINATION

General Appearance.—A young man of twenty-nine, lying quietly in bed, not apparently acutely ill. Quite well developed and nourished, except for his hands and forearms which are markedly wasted. There is a moderate posterior spinal curvature in the upper dorsal region. His chest has an abnormal anteroposterior diameter and the lower end of the sternum and ribs protrude rather prominently. There are constant fibrillary contractions of the muscles of his arms, the upper portion of his back, and at times on the left side of his face. They are most marked and constantly present in the right triceps muscle.

Skin.—This is everywhere soft and smooth, with no trophic disturbances.

Head.—The cranium is large and symmetrical; there are no evident abnormalities. The right side of the face seems smoother than the left and to have less expression. There is no tenderness of the scalp or of any portion of the head.

Eyes.—The right palpebral fissure is slightly larger than the left and the left eye is slightly less prominent. The left pupil is smaller than the right. Both are regular and react to light and on accommodation. On looking to the right or left there is a rotary nystagmus. The conjunctivæ and scleræ are clear.

Ears and Nose are negative. The ear-drums appear normal.

Mouth.—The lips are normal. The right side of the tongue is atrophied and the surface much wrinkled. There is a slight continuous fibrillary tremor of this portion of the tongue. The uvula is in the mid-line. The tonsils are very small and not inflamed. The teeth are rather dirty with a moderate pyorrhea, but no evident caries.

Chest is prominent, rather barrel-shaped. It moves symmetrically with respiration.

Lungs.—Resonance normal. The breath

and voice sounds are entirely normal throughout. There are no râles.

Heart.—The apex beat is not seen or felt. The outline is normal. The sounds are rather muffled but are of normal character and there are no murmurs.

Abdomen is symmetrical. No tenderness, masses or signs of fluid.

Liver and Spleen are not felt.

External Genitalia appear normal.

Superficial Lymph Nodes.—The glands in both groins are slightly palpable, but are not tender. There are no other enlarged glands.

Extremities.—The muscles of both forearms and hands are atrophied, the flexors probably a little more than the extensors. The thenar and hypothenar eminences of both hands are practically gone. The hand appears flattened out and extends at the wrist whenever he endeavors to flex the fingers. He has very little strength in either hand. The first finger of the right hand is flexed at a right angle at the second joint due to an old injury in which the extensor tendon was cut. There are two small, bluish swellings on the posterior surface of the right forearm. One of them is about 3 cm. long by 2 cm. wide and is slightly elevated above the surface. It appears to be a subcutaneous hematoma. The flexion and extension of the arms is fairly strong and about equal on the two sides, but there is evident atrophy of the muscles of both forearms. There is also some evident muscular atrophy of the right shoulder girdle. The right pectoral is much smaller than the left. The lower extremities are negative. There is no edema or tremor.

Joints.—Movement of all joints is practically normal, but there is everywhere present a slight crepitus.

Temperature.—98.8°.

Pulse.—78.

Respiration.—18.

Weight.—167¼ pounds.

LABORATORY EXAMINATIONS

4/28/19. <i>Blood Examination:</i>	
Red Blood cells	5,200,000
Hemoglobin	95%
White blood cells	12,600
Polymorphonuclear leucocytes	71%
Large lymphocytes	5%
Small lymphocytes	24%
Eosinophiles	0%
4/29/19. <i>Blood Sugar:</i>	
.86 gm. per litre.	
4/29/19. <i>Blood Wassermann:</i>	
Alcoholic antigen	negative
Cholesterin antigen	negative
4/30/19. <i>Lumbar Puncture: Spinal Fluid:</i>	
Pressure	Slightly increased: about 120 drops per minute
Appearance	Transparent and perfectly clear
Cell count	4 lymphocytes
Globulin	Negative
Culture	No growth
Wassermann	2 c. c. 1 c. c. 0.6 c. c. .2 c. c.
Alcohol	Negative Negative Negative Negative
Cholesterin	Negative Negative Negative Negative
4/30/19. <i>X-ray Examination of Spine:</i>	
There is no indication of disease of the cervical or dorsal vertebrae. There is a deviation of the outer dorsal spine toward the right, but this may be due to posture. The vertebral bodies all appear normal.	
4/30/19. <i>Urine Examination:</i>	
Specific gravity	1.018
Reaction	Acid
Color	Amber
Turbidity	Slight
Precipitate	None
Albumin	None
Glucose	None
Microscopical examination	A few amorphous urates. No casts, crystals or cells.
4/30/19. <i>X-ray Examination of Arms and Hands:</i>	
No evidence of rarefying osteitis or any joint abnormality.	

NEUROLOGICAL EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Attitudes.*

—The patient stands with his head bent slightly forward. There is a round posterior curvature of the spine in the upper dorsal region. The chest is prominent and has an increased anteroposterior diameter. The lower end of the sternum protrudes prominently. There is evident atrophy of some of the muscles in the right shoulder group which gives rise to an asymmetry of the right shoulder and right portion of the chest. The forearms and hands dangle from the elbows in a peculiar, helpless, skeleton-like fashion. The abdomen and lower limbs appear entirely normal.

Gait.—Normal.

Coordination.—Coordination of the equilibratory and non-equilibratory type normal.

Skilled Acts.—There is no dyspraxia or speech disorder. His writing is very poor due to an inability to hold a pen firmly and to his muscular weakness.

Reflexes:

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Jaw	Active	Active
Pectoral	Absent	Absent
Biceps	Absent	Absent
Triceps	Absent	Absent
Radial	Absent	Absent
Ulnar	Absent	Absent
Wrist	Absent	Absent
Suprapatellar	Very active	Very active
	With transient clonus	
Patellar	Very active	Very active
	Possibly a little more marked; clonus present	
Achilles	Active	Active
<i>Superficial</i>		
Ciliospinal	Present	Absent
Supra-umbilical	Absent	Absent
Suprapubic	Absent	Absent
Upper lateral abdominal	Absent	Absent
Lower lateral abdominal	Absent	Absent
Cremasteric	Absent	Absent
Babinski	Present	Present
Chaddock	Absent	Absent
Oppenheim	Absent	Absent
Gordon	Absent	Absent
Schaefer	Absent	Absent

Abnormal Involuntary Movements.—Fibrillary tremors are constantly present in the right triceps and at times are visible in the left triceps, biceps, deltoid, pectoralis major, also in the right biceps, deltoid, latissimus dorsi, and occasionally in the muscles of each forearm. None are noted in the muscles of either hand.

Muscle Strength.—Rotation of the head and forward and backward movements seem to be normal and equal on the two sides. *Shoulders:* Elevation of the shoulders appears normal. *Upper Arm:* Abduction slightly weaker on the right, though the patient is able to raise his arm above his head easily. Adduction of shoulders is less marked on the right side because of weakness of the right pectoral muscle. *Forearm:* Flexion is weak on both right and left, probably more on the left.

Hand. Dorsal flexion very weak on both sides, most marked on the left. Ventral flexion very weak at the metacarpophalangeal joint, somewhat better in the terminal phalanges. He is unable to close the left hand and can close the right one only with marked dorsal flexion. Abduction and adduction of the fingers are very weak, a little stronger on the right side. *Trunk:* Patient can rise from the dorsal to the sitting position very easily. The rotation of the trunk is apparently normal. The movements of the lower extremities are normal and equal on the two sides.

Muscle Status. — There is marked

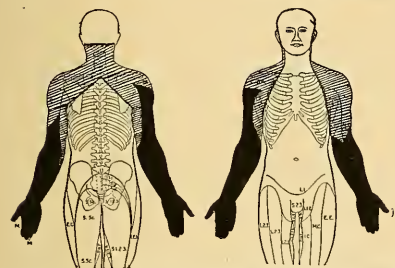


FIG. 4. DARK AREA—LOSS OF TEMPERATURE SENSE. SHADED AREA—TEMPERATURE DISCRIMINATION NOT ACUTE.

diminution in the volume of all the intrinsic muscles of each hand and of each forearm, the extensors, however, being a little less wasted than the flexors on each side. The biceps and triceps on each side are partially atrophied and flabby. There is diminution in the volume of the muscles of the right shoulder girdle both anteriorly and posteriorly. The affected muscles on both sides are soft, flabby and hypotonic.

Abnormal Associated Movements.—No observations.

Nerve Status.—No record.

GENERAL SENSORY. Touch.—Entirely normal. *Pain:* Hypalgesia over both forearms and the outer portion of the right arm. There is no absolute analgesia and even over the affected areas he can almost invariably discriminate between the point of a pin and the head. *Temperature:* There

is absence of the temperature sense in the areas described as hypalgesic. There is also decreased ability to discriminate between hot and cold over both shoulders, front and back, the back and lateral portions of the neck, and over a portion of the pectoral and axillary regions on the right side. Over the affected areas he does not seem to perceive either temperature better than the other. *Vibratory:* Normal over both extremities and trunk. *Pressure:* Normal. *Muscle-tendon:* Normal. *Stereognosis:* Normal.

CRANIAL NERVES. Olfactory and Nose.—The sense of smell is normal on each side.

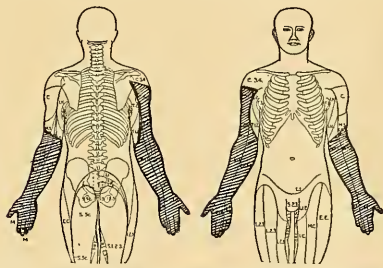


FIG. 5. SHADED AREA—DIMINUTION OF PAIN SENSE.

Optic and Oculomotor Apparatus.—*Visual Acuity:* 20/20 in each eye. *Perimetry:* Normal to the hand test. *Eye Grounds:* Normal. *Pupils:* Round and circular and regular. The right is larger than the left, measuring 4 mm., the left 3 mm. They both react actively to light, convergence and accommodation; the left, however, dilates imperfectly to dark. The left palpebral fissure is smaller than the right, measuring 7 mm., the right 9 mm. There is enophthalmos of the left eye, the position of the left eye being 17 mm. with the exophthalmometer while the right is 18 mm. There is no strabismus or other ocular deviation. The conjugated eye movements are normal. There is a slow rotary nystagmus in each eye on looking to the left. There is a horizontal and rotary nystagmus in each eye on

looking to the right, the slow movement being toward the left.

The Acoustic Nerve and Ear.—A watch is heard 18 inches from the right ear and 24 inches from the left ear. Air conduction is greater than bone on each side.

Trigeminus Nerve and Mouth.—All movements of the jaw are normal. Sensation is normal in the areas supplied by the trigeminal nerve. Both corneal reflexes are normal.

Facial and Face.—There is slight asymmetry of the face, the left side being smoother and flatter than the right. This is most marked around the mouth. There is, however, no evident weakness, forced facial expression being symmetrical. There is an absence of the secretion of sweat on the left half of the face.

Glossopharyngeus and Vagus Nerves, Pharynx and Larynx.—The uvula is in the median position. The palatal and pharyngeal reflexes are normal. There is no abnormality noted in the act of swallowing. The phonation and quality of the voice are normal.

Spinal Accessory Nerve.—Movements of the head and shoulders are apparently normal.

Hypoglossus Nerve and Tongue.—The tongue is protruded slightly to the right of the mid-line. Movements produced by the right half of the tongue are less marked than those on the left. The right

half of the tongue is wrinkled and atrophied with constant fibrillary tremors. There is a slight oozing of blood from the dorsal surface of the atrophied side.

Mental Status.—Apparently normal.

Cranial Morphology.—Normal.

Systemic.—There are no trophic disturbances in the skin, subcutaneous tissue, bones or joints.

CONCLUSIONS

The chief features of interest in this case are:

1. The absence of true analgesia, though there is a loss of the temperature sense and evidence of an extensive lesion extending from the region of the seventh nerve nucleus in the bulb to the first or second dorsal segment of the cord.
2. The absence of trophic disturbances in the skin, subcutaneous tissue, bones or joints.
3. The presence of the subcutaneous hematmata over the wasted muscles, which, it is believed, are probably the result of slow oozing spontaneous hemorrhages similar to those observed over the atrophied portion of the tongue.

PERSISTENT HEADACHES, IRRITABILITY AND STATE OF DEPRESSION DUE TO THE RETENTION IN THE SCALP FOR SIX MONTHS OF A FOREIGN BODY

BY LOUIS S. ARONSON, M.D.

NEW YORK CITY

S. S., age twelve, first came under my observation when he was four years old. There was a rather severe rheumatic endocarditis, with loud systolic mitral murmurs. The family lived on the fifth floor of a tenement in a very congested district and as a result the boy was kept either on the street too much, or indoors too long, the climbing of the stairs being a hardship for him on account of his cardiac condition. For this reason the parents removed to the northern outskirts of the city, to a life simulating that of the open country. Up to a year or so ago I saw the lad about once or twice a year for trivial matters. On account of his weak heart the boy was very badly reared. His parents knew nothing of pedagogical training and feared to coerce him by punishment. As a result he grew up in the street, and although very young associated with older boys, was precocious, bad and rapidly deteriorating. He had a similar mental makeup. He was reticent about injuries sustained, and would tell his parents that he was able to take his own revenge on those that hit him. He was soon known as the worst boy in the neighborhood. His habitat was the street and he came into the house for food and sleep only. The neighborhood was frequently a miniature battle ground, with sticks, stones and bottles used as weapons. That is how he so frequently became injured. I counted over forty ecchymoses distributed over all parts of his body about a year ago. He usually had one black and blue eye; occasionally both were discolored. He seemed quite obtuse to pain.

On Nov. 1, 1918, his parents brought him to me for a mental examination. They said that for six months the boy had been

suffering with very severe constant, in fact daily, headaches, that seemed to have effected a change in his mentality. He seemed to be much more irritable, but less ferocious, and for the first time in his life complained of pain. He would come from school and remain in the house, which was not his custom. He did not care for the streets, was quiet and morose, and unusually drowsy. He would fall asleep in very peculiar attitudes. He would rest his forehead on his elbows. He would sleep on his abdomen in bed, or leaving his body in bed would place his head on the window sill. The mother interpreted this as a grave sign and took him to numerous doctors and clinics. He was apparently carefully examined. They examined his urine, and took a Wassermann blood test. He was given eye-glasses, and had ingested a great deal of medicine with no relief.

He cried in my office and refused to let me examine him, nor would he state anything about his history or the character of his pain. I drew him towards myself by placing my palm against his occiput, and he uttered a loud cry. At first I was at a loss to know whether this was indicative of fear, refusal, or pain. I was about to look for some of the nodules described by Auerbach, as I knew the boy to be so thoroughly rheumatic. To my surprise closer examination revealed the fact that there was an area a little larger than a silver dollar at about the region of his external occipital protuberance, which was very tender and quite edematous. At about its center was a small purplish papule, about the size of a grape seed. It looked to me at first like a small nevus, but on palpating the edematous area a little, the raspberry-like mass burst, a little

serosanguinous fluid escaped, and with that there protruded a small piece, about $\frac{1}{8}$ inch of foreign substance. This I seized with a small forceps and extracted. It was a piece of wood, about an inch long, and the size and shape of half an ordinary square toothpick. After shaving the region, and under a local anesthetic, I made an incision of about one inch down the center of the edematous mass, and found a second piece of wood thinner and about one-third the size of the first, lying in a position almost parallel to it. There was also considerable débris consisting of old blood clot and muco-serous secretion, possibly with some pus. I painted the cavity with iodine, inserted a small piece of gauze drain, and applied a Burrow's solution dressing. After several such dressings the boy's wound healed. His headaches have entirely ceased; he is not so irritable

or drowsy; his conduct is a good deal better, though I do not expect any real change in his behavior. At any rate it is too early now to prognosticate on that.

The patient subsequently admitted that he had been struck on the back of his head by another boy six months previously, the weapon being part of the cover of a wooden soap box. That this was so was corroborated by the mother, who recalled that the patient went out on the street, had a fight and came in with a bleeding scalp. The mother sent him to a corner drug store for first aid. This the druggist rendered. It consisted in placing a small piece of cotton soaked in peroxide against the cut. The bleeding ceased at once, and as the lad was not of the kind to complain, the wound healed, and he himself did not know that his headaches were related to his tender scalp.

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THE FUNCTIONS OF A MENTAL CLINIC *

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It is difficult to escape the feeling that the task of bringing before this conference for discussion the subject indicated by the title of this paper has something in it very much akin to the proverbially superfluous task of carrying coals to Newcastle.

I am, nevertheless, grateful for the opportunity, because of a real personal need for a better orientation in this subject, if for no other reason.

It is doubtful whether any discussion of the functions of a mental clinic can lead to constructive results without taking account from the very beginning of the fundamental differences that exist between psychiatry and other branches of medicine, of the many respects in which the disorders and difficulties dealt with in a mental clinic differ from those which one encounters in the general medical or surgical dispensaries, or even in a strictly neurological clinic.

The latter, especially the medical and surgical dispensaries, deal essentially with disease entities and symptoms which, though more or less incapacitating to the individual, create problems in the main, and in the majority of instances, confined to the individual himself.

A dispensary patient suffering from a strictly medical or surgical disability, though incapacitated to that extent, is still able as a rule to maintain an efficient relationship with his fellow-men. A woman might be coming to the clinic for weeks to

be treated for some kidney or gastric difficulty, but need not on this account lose her capacity to conduct her household affairs, or to rear her children properly, or be all that she ought to be to her husband and neighbors.

A man similarly affected need not become on this account an annoyance or a menace to his shop or office associates. Not so with psychiatric disabilities. Here one deals almost always with "situations"; and although the specific symptom or disease still plays the central rôle in the picture, it by no means tells the entire story. The problems created by psychiatric disabilities are never, because of the very nature of these disabilities, strictly confined to the patient himself. One does not deal here with a disturbed function of some one or other organ of the body, but with a disordered functioning of the personality as a whole, which carries with it inevitably a disturbed relationship between the individual and his environment; and the problems which this disturbed relationship creates may find a reverberation in the school, the home, the shop and the market. Furthermore, while it is always to the community's advantage to give the subject of disease among its members due and constant attention, when it concerns psychiatric disabilities this becomes especially imperative, since what is in one instance merely a social disability, due to loss of productive capacity, may easily become in the

* Read before a Staff Conference of the Neurological Department, Vanderbilt Clinic.

other instance a real social menace. Thus the relation of the psychiatric dispensary to the community is a most intimate and important one, since it deals largely with disordered social relationships.

This is, of course, trite matter and well known to all of us, but it merits restatement, if for no other reason than to recall the very specialized approach which a recognition of these facts implies. That the approach here differs essentially from what might be suitable in dealing with other non-psychiatric issues of a dispensary is perfectly obvious, but it is not always so obvious that this difference affects every step in the procedure, the aims and atmosphere of the clinic, the relationship between the patient and the physician, the time element involved, and finally, what is most important, the frank and straightforward recognition of the fact that in the dispensary treatment of psychiatric conditions, more so than in any other field of medicine, we are almost helpless without the aid of efficient social service. An exclusively medical therapeutic approach can never satisfy the needs of situations where the medical issues are so closely interwoven with significant social-economic issues as is the case in the maladjustments constituting the bulk of psychiatric practice. There is needed here a kind of extra-medical aid which only the trained social worker can supply.

The central aim in psychiatric practice, as in all medical practice, is, of course, the helping of the patient through an alleviation of his symptoms or through an absolute cure. But indications and methods are in the main clear-cut in non-psychiatric problems, while they are usually very complicated in dealing with disorders of the personality. Treatment here does not mean simply the correction of some isolated function, but frequently involves a reshaping of the entire personality, a correction of life-long habits of living and acting, a modification of attitudes towards life. The overt condition which may have led the patient to seek help, may be the outgrowth

of fundamental biologic maladaptations, of faulty adjustments, of immature or distorted methods of meeting the complex situations of life. Furthermore, treatment here frequently involves paying attention to some other members of the patient's family, since his own faulty adaptations may be dependent very much upon faulty attitudes of those with whom he has to live. This is particularly true in dealing with maladjustments in children.

A five-year-old boy was referred for treatment on account of excessive and open masturbation, nervousness and pugnacity. The boy was found to be a very neurotic, considerably defective, poorly nourished and physically neglected child. Great difficulty was experienced in examining him since he could not be separated from his mother for any length of time, and the procedure affected her if anything more than the child. She cried, wrung her hands and stood behind the clinic door ready for whatever emergency might arise. From the very first it was obvious that she needed treatment as much as the child and that she was contributing very materially towards the youngster's tyrannization of the household. She is herself a highly neurotic and emotionally unstable woman, cries on the slightest provocation, and great difficulty is encountered in fixing her attention. She is very fond of her children and hovers over them with animal-like devotion. She was herself totally unfit to meet life's fundamental problems adequately, and it is out of the question to expect her to deal intelligently with her boy's problems without much education. On her wedding night, when confronted with the concrete facts of conjugal relationship, she ran away from her husband, and it was only after much assurance by her mother that all men were alike and that she must face certain fundamental facts that she returned to live with him.

Her married life has not been very happy, and her attitude toward her children became conditioned in a particularly faulty way through the accidental death of

one of them. Our patient has always been a nervous child, has had chicken pox and whooping cough and has at present enlarged tonsils and adenoids. He has masturbated continuously since he was one and a half years old, would rub himself against the furniture, on the floor, or handle himself manually. He does it without apparent embarrassment, in the open, at school, at home and on the street. He is very irritable and quarrelsome, does not get on well with the other children on the street and practically tyrannizes the household. He is excitable, unstable emotionally, at times hypomaniac, and is restless and wakeful nights. No bed wetting. He sleeps in his own crib in the same room with his parents and sister. He is given tea and coffee at least once a day, and practically everything else he desires in the way of food. He entered school last autumn, but does not get along very well because of his quarrelsome and destructive disposition. These difficulties of conduct have been handled by the parents in a very unintelligent manner, although they are pathetically anxious to do the best for their children. Their conception of this, however, is to let the boy have his own way in everything. They indulge in much petting and fondling of the children, and until our patient was four and a half years old his mother wheeled him about in a go-cart. She never allows him out of her sight in his play on the street, and when he gets into any difficulty with the other boys she immediately intervenes. She brings both of her children to school and calls for them, and makes a great hubbub if they are even spoken to roughly. The teacher and the vice-principal state "the woman has no discipline over her children and fondles them in an animal-like fashion." Of late she has kept the boy away from school altogether.

With this sort of background one can imagine how hopeless it would be to confine treatment to the child. The therapeutic indications here, as we saw them, were in the main, first, improvement of the child's hy-

giene in the matter of diet and bathing; second, a training of the mother in a more intelligent care of the boy. She was advised to cut out overstimulating foods from the child's diet, such as tea, coffee, excessive meat and candy, and to give him prolonged hot baths evenings before retiring. She was urged to allow the child a little more freedom in his play activities and contacts with other children, and to wean herself from the excessive need for emotional gratification which she was getting from constant fondling of the boy. She was advised to encourage him to fight his own battles instead of doing this for him, and to appeal to his desire to grow up to fine manhood instead of threatening him with going crazy, as she has been doing in her efforts to correct his habits of self-abuse. She was to return the boy to school as soon as possible and to cooperate better with the teacher in the training of the child.

These were some of the things that suggested themselves in the treatment of this boy. With all her serious limitations the woman tried her best to follow out instructions, and on his second appearance at the clinic, about three weeks later, the boy looked very much better physically and was very much improved in behavior. He had given up his habit of self-abuse, had returned to school where he was getting on better, and was much more cooperative and accessible at the clinic. Much still remains to be done in getting this ignorant, overburdened mother to appreciate fully the tasks before her in the rearing of this neurotic child.

This case illustrates the point we are trying to make about the necessity of reaching out in our treatment beyond the patient himself, and the important rôle which attention to what might appear to be very small and insignificant details plays in the treatment of misconduct. The illuminating and helpful picture of the setting in which this child lives was furnished by the social worker; and much of what we may ultimately accomplish in the present case will depend upon the extent to which

the social worker will be able to modify the situation along the lines of medical advice.

But it is not only in dealing with children that one meets with unhealthy parent-child relationships demanding serious attention if anything constructive is to be accomplished.

A girl of nineteen was referred for treatment for what had been tentatively diagnosed as a post-influenzal endocrinous disturbance. Following an attack of influenza in October last, she developed various neurotic symptoms, such as sinking sensations, especially when reclining, a sensation of falling downward; things danced before her eyes, and she had a feeling as if her eyelids wanted to close all the time. She had frequent hysterical attacks, dreaded to go to sleep at night, staying up frequently until midnight with her mother, from whose company she derived much comfort. She also suffered from anorexia, epigastric distress, and constipation—a complex of symptoms which had invalidated her to the extent of having kept her from work for months.

The physical examination disclosed nothing tangible to justify the diagnosis of an endocrinopathy, though the girl had a slightly enlarged thyroid; but a better acquaintance with her brought to light some very fundamental biological difficulties in her personality development which demanded attention if she was to become an efficient social being. She is the daughter of a lazy, worthless father, whom the mother finally deserted in Russia, coming to America when the patient was about sixteen months old. The mother and daughter lived together in the home of the former's married sister who has been insane for many years but is kept at home. The mother supported the child and herself by doing midwifery, and the two grew to be very dependent upon each other for their happiness. At the time of her visit to the clinic, although nineteen years of age, the girl was still sleeping with her mother, the mother still selected and bought her clothes for her, and was expected to select her hus-

band for her. She treated the daughter in practically everything as though she were a little child, spoiling her in many ways and giving her no opportunity for developing a sense of responsibility or any initiative. The girl always turns to the mother for her opinion, and the two feel that they have only each other in the world.

The girl attended school until the age of fourteen, reaching the 7A grade, since which time she has worked in stores, earning at the time of her illness, in the fall of 1918, \$9 per week as saleslady in a mediocre store. She had a good reputation there and tried to work after her influenza attack, but could not endure the strain and had to leave.

We found the girl to be extremely immature and childish in her makeup. She recited in a whining, plaintive manner her ailments, dwelling particularly upon her gastric distress and sinking attacks. She professed a desire to go to work, but was sure she couldn't stand it since it was absolutely impossible for her to eat outside of her home; in fact, she could eat no food except that prepared by "mamma." Felt especially keenly her need of mother's companionship and comforting, following a disastrous ending of her first and only love affair. For about a year she kept company with a young man considerably above her station in life in wealth and otherwise, and although he called on her frequently, he never entered the home, but met her on the doorstep. He was apparently conscious of the disparity in the social standing of the two, as was she. He was in the habit of making appointments with her over the telephone, then breaking them uncereemoniously while she would be standing at the window until midnight, crying. She was very much in love with him, and although she tried to make the best of it when he finally jilted her and married a rich girl, for weeks afterward she did nothing but cry every evening. While in this disturbed state, and about a month after her lover married, she had the attack of influenza which reduced her very much physically

and tended to aggravate further her mental burdens. The fact that the girl was practically devoid of constructive attachments and interests outside of her employment and her mother, that she had but few girl friends, had never participated in any club or group activities, and very seldom spent her evenings away from her mother, did not help her to adjust herself to her disappointment.

During her illness she spent practically all the time at home with her mother, who had to give up her work too, so that the economic problem was becoming an acute one.

This was the situation confronting us when the girl came to our attention. Whatever strictly medical therapeutics was indicated in the case, it seemed obvious that a good deal of attention was required in the matter of reshaping the attitude and mode of life of both mother and daughter. One could not ignore the fundamentally faulty relationship between the two, the manner in which this led to an absolute dependence of the daughter upon the mother, and to the latter's retention of a kind of fondness and maternal attitude towards this nineteen-year-old girl that one would expect towards an infant. The two had retained ideals of marriage and social relationships which were wholly out of keeping with American life and customs.

The record of the social service procedure which was required to meet this problem is too extensive for reproduction here. It involved efforts at weaning the girl from the mother through the development of new interests and new means for self-expression. We had to get her interested in girls' clubs, settlement activities, dancing, etc. It involved searching for the kind of work which really held the girl's interest and which might possibly gratify a latent ambition to work in a more refined place than formerly. It meant a persistent arguing in favor of the virtue of a little more independence of thought and action in choosing recreational and other outlets and in favor of healthier ideals about marriage and personal hy-

giene. Finally it required the extremely difficult task of getting the mother, who persisted in balking our efforts, to realize the girl's absolute need of a chance to grow to maturity and greater independence. The girl came to us on February 11th, and on March 27th she wrote the social worker that she was better and happier and at work. Incidentally, with a shifting of her interest toward more vital and fundamental issues, the various somatic complaints have taken care of themselves.

The foregoing two case reports suggest briefly what the aims of a mental clinic might be as regards individual therapeutics. But the aims of a psychiatric clinic should go considerably beyond this. The community at large is appreciating more and more fully how closely related are the various community problems which are reflected in such institutions as the truant, probationary and parental schools, juvenile and adult courts, prisons and reformatories, and even the ordinary relief agencies, to the mental health and makeup of the individual members of society.

It is but natural that they should look upon the mental clinic as a community institution which ought to aid them materially in the solution of these various problems, and this ought to be an attractive aim of every such clinic. One cannot enter here into a detailed discussion of ways and means, but it is truly lamentable how little initiative the average psychiatrist manifests towards making his influence felt in these directions.

It would be truly regrettable if the psychiatric clinic were not to contribute its share towards the spreading and popularization of such mental hygiene principles as have been found to be practical and useful. It might do this through its extensive and varied contacts with the community, but especially so through being fully alive to the training possibilities which such a clinic offers for social as well as medical students in the management of the concrete issues which come before it.

These wider aims of the mental clinic are

to my mind just as vital as is the primary aim of bringing relief to the individual patient.

The second point we touched upon in the beginning of this paper was the atmosphere of the clinic. If our enterprise is to contribute truly to the promotion of human happiness and freedom from the harassing states of mental trouble, we must pay considerably more attention than has been the case in the past to the physical and mental atmosphere of the clinic. Above all we must take our work more seriously and must persistently train ourselves for a truly altruistic tolerance toward human foibles and follies. We must leave moralizing to those from whom people expect lessons in morality. This need not, of course, interfere with our opportunities for constructive guidance in living, thinking and acting.

I noticed to-day an article in the *Journal of the American Institute of Criminal Law and Criminology* entitled The Experience of a Psychiatric Missionary in the Criminal Courts. This is significantly symbolic of what I have in mind.

A woman aged thirty-five was referred for treatment on account of distressing thoughts, anxiety, fear, and enlargement of the thyroid. The record stated that the woman had influenza and pneumonia in October, 1918. Towards the end of that month while going to bed, she fell on the floor and had an intense apprehension that she was going to die. She was in bed two months, the attacks would recur about twice a week, were of varying intensity, always of the nature of a dread and fear of death and accompanied by cardiac palpitation. She was depressed and lachrymose, and a few nights after Christmas developed the fear that she would kill her child and that she had to commit suicide. She could not trust herself to be alone at home and her husband had to give up his work and stay with her constantly.

When we first saw her the woman was in a most intense state of anxiety, fear and emotional tension, so much so that we considered it unwise and dangerous to delve into her symptoms. Her pupils were di-

lated, she was constantly wringing her hands, and would frequently glance about the room in an apprehensive manner. She wept readily and profusely and repeated anxiously, "What have I done to deserve this, to want to kill my only boy and to destroy myself?" After much assurance we encouraged her to talk freely, but nothing coherent and helpful came out beyond the statement that her head was full of nasty, horrible thoughts, and that she could not talk about them. We allowed her to go after a brief consultation. Upon leaving she expressed herself to the social worker that she hated the doctor and did not think she would come back, or words to that effect. She returned, however, two days later, very much quieter and distinctly better and told with a little encouragement the following story: She had been married for about eleven years and throughout this time had been tortured by a conflict which arose out of a desire to tell her husband how she had been seduced and had carried out an abortion before her marriage, and a fear of the effect this might have upon their relationship. At her menstrual periods she was particularly unhappy, would weep for hours at a time and gradually developed the feeling that her husband suspected something was wrong, but she could never get herself to face the situation squarely. While convalescing last fall from the attack of influenza, she got the notion that she wanted a Bible and asked her husband to buy a Swedish edition. He did so, and the first time she opened it she came across the phrase "flesh of my flesh" and the entire early sexual experience and subsequent abortion came back to her with an intense and overwhelming force. She threw the Bible away in horror and the compulsive thoughts of suicide and infanticide took possession of her. After the first interview she went home, made a clean breast of the whole affair to her husband and as he fortunately took a right attitude towards the matter and assured her that it could not in any way impair his affection for her, she felt immensely relieved.

She came back only once more and dis-

cussed the details of her early experience, some of which involved perverse demands upon her by her lover, which she had not discussed with her husband. We assured her that there was not much significance in this and that she might just as well leave those things unsaid. As she did not return to the clinic we addressed a letter to her husband inquiring about her health and suggesting that we would like to see her at the clinic. In response to this he telephoned to the clinic on February 27th, only sixteen days after we first saw the case, that his wife was very much better, had gone to Lakewood, and because of this had not come to the clinic. On March 10th the social worker called at the home and met the husband on the doorstep. He said that his wife had returned from Lakewood, was very much better, "two hundred per cent better" as he put it, than when she first came to the clinic; was taking long walks, going to dances and movies. He was planning to go to work the following week. He said his wife had a great dread of returning to the clinic, since she feared it might stir up her old thoughts, etc., and he very apologetically begged that it be not insisted upon, promising to bring her back should she show any signs of a return of the difficulty.

The woman is, of course, far from absolutely well, but the improvement which did take place is really phenomenal. It would be difficult to explain, however, if one were called upon to do so, just how the clinic consultation brought about this change in her condition, for one cannot speak here of anything even approaching psychoanalysis. I am certain, however, that had the physician's attitude toward her betrayed any signs of censure or of a desire to moralize the result would have been far less satisfactory.

The physician is there to understand and not to censure, to assist through methods more agreeable than moral lectures. Dwelling upon these points concerning the importance of the atmosphere of the clinic in dealing with mental patients is fully justified when one keeps in mind the conditions

under which the psychiatrist has to work in the average clinic.

One ardently hopes that some way may be found for doing away with the horribly long waits these sick in mind must endure, and if this is unavoidable, that some proper provisions may be secured for occupying the time while waiting. The classes in occupational therapy, such as basket making and rug weaving, used at some dispensaries, and a children's playroom, suggest themselves as inexpensive and very effective means. Other methods might well be experimented with in the greater humanization of the average dispensary.

It would take us altogether too far afield to enter into a detailed discussion of the relationship that is to be maintained between the physician and the psychiatric patient. But it is important to keep in mind the time element involved in dealing with these cases. While the initial examination is as time consuming in the neurological case as it is in the psychiatric case, the subsequent consultations that are needed, if one is to accomplish anything worth while in the treatment of mental patients, is even more time consuming than the initial examination. The following case is illustrative of this point. With your permission I will present this case in accordance with a record scheme which we are using in the instruction of the psychiatric social worker.

The Problem: A Russian Jewish immigrant, aged thirty-two years, was referred for treatment for a condition suggestive of hysteria. Since the middle of November, 1918, he had not been working, because of a complaint that he could not see; has been practically mute except that on rare occasions he can be provoked to talk; has been in a rigid, listless attitude, wholly out of touch with his environment, practically sitting in one place all the time.

The economic situation of the man's family is becoming an acute one, since his earnings were the only source of income of the family of five. His wife, who is very much worried and distressed, had been advised by rich relatives to send him to a

sanatorium, place the children out and go to work, but would not listen to such a solution.

Physical Status: He is well built and well developed physically, general state of nutrition good. Attitude and gait very rigid. In walking the body moves as though the joints were fixed, head is tilted backward and is held in the attitude of a blind man. Neurological examination carried out with difficulty revealed no abnormalities. Ocular movements, pupillary reflexes and visual fields were normal. Patient stated that he could see to walk but nothing else.

Mental Status: Feb. 6, 1919. Patient entered the examining room in a constrained, stiff manner, took the proffered chair upon request and sat motionless with a fixed gaze upon the examiner. Occasionally a smile broke the immobility of the facial muscles. He replied fairly freely to questions not directly concerned with his present illness, but as soon as this subject was touched he became practically mute and resorted to the phrase, "I don't remember nothing."

Replies were always to the point, but were given in a markedly retarded manner and low tone of voice. Responses were emotionally colorless for the most part. He was accurately oriented for place and person, but slightly uncertain about time. Knew the day of the week but not the date. Attention was gained readily and held without difficulty as long as subject of present illness was not touched. When this became the topic of inquiry he had to be urged long and energetically before he replied. General memory was good for remote and recent events, but he insisted that he had a complete amnesia covering the entire period during which he was employed by his cousin and in connection with which employment his illness developed. The precise limits of this amnesia could not be ascertained.

No delusions or hallucinations could be elicited. He insisted that he was practically blind, that he could barely see to walk, could not be made to read. No feeling of

being controlled by outside influence could be elicited, and although general musculature was rigid he showed nothing further typical of a catatonic state. Has been more or less impotent sexually since illness and is emotionally depressed over this. General emotional tone that of mild anxiety and depression. He complained of poor appetite and insomnia and insisted that he was not getting better.

Medical and Social History: The patient, the issue of plain Polish-Jewish stock, was born in a small Polish town, and is the oldest of ten children. Nothing of significance has been ascertained in his heredity, and birth and infancy are said to have been normal. His father died when patient was between four and five years of age, and he spent his boyhood and adolescence under the influence of a step-father with whom he seems, however, to have gotten along very well. School life, which was chiefly confined to sectarian subjects, terminated at twelve because he was obliged to devote his time to assisting his parents in the conduct of a small grocery store. Work was hard, hours long, but he was well able to adjust himself to the requirements. At eighteen he was drafted into the Russian Army; upon his release he set up in business for himself, and at twenty-three married.

Life continued quite uneventful until 1910, when a well-to-do American cousin visited them and persuaded the patient to immigrate to the United States, whither his wife followed him a year later. The family got on very well here. Marital relations are said to have been very satisfactory, he has always been a devoted husband and a good provider. All his interests and affections centered around the home, he never went out without his family, and assisted his wife with much of her housework. He worked steadily and faithfully, frequently overtime, and managed to gratify their simple wants.

Home hygiene, however, was not of the best, probably largely because the people with whom they associated and from whom

they got their patterns of Jewish immigrant life, lived no better. The house, while kept very clean, is crowded, the family of five and a cousin boarder occupying a three-room apartment, and the man, wife and three children sleeping in one room about 8' x 8' in size.

In September, 1918, the patient's brother was married in the patient's house to a woman whom the patient intensely disliked. At this wedding the rich cousin mentioned above offered the patient a position in his watch factory. The offer came about through the suggestion of the patient's wife that he might do something for her husband.

The patient himself was unwilling to go, was afraid to change jobs and constantly worried, according to the brother's statement, that he was not earning his salary and wondered why the cousin should be so good to him all of a sudden. The wife's ambitions, however, prevailed, and his acceptance of the position followed. His work consisted in placing crystals on watches by the aid of a gauge with very fine figures. He also did various odd jobs about the place. After about six weeks at this new job, he accidentally struck his head against an iron instrument which caused a freely bleeding scalp wound requiring a bandage for over a week. A girl fellow-worker stated that he changed in his behavior decidedly after this. Instead of being gay and cheerful he became silent and moody, would weep in the midst of his work, saying that he was afraid he would lose his job. A few days before his complete breakdown he complained of his eyes. He stated to members of his family that he feared he would lose his position on account of his bad eyesight.

He consulted an optometrist, who prescribed glasses, but his eyesight did not improve and the optometrician told him that he would have to give up his work or go blind. He came home greatly discouraged, cried, felt he had failed, but the next morning started for work as usual. He turned back, however, came home, sat down and

wept because he was going blind. He rapidly grew worse, more rigid in attitude and bearing, more mute, until he finally assumed a practically immovable attitude, did not notice the children however noisy they became, and refused to speak to his wife. A nerve specialist whom they consulted at this time advised sanatorium treatment, but wife refused to listen to this and instead took him to Lakewood where they remained three weeks.

When his brother visited him here he refused to talk to him. Patient's wife was lying on the floor weeping and reproaching him bitterly for not supporting his family, but not a word could be gotten out of him.

After they returned to New York the brother took the patient to a Christian Science healer who prayed with him, whereupon he became very angry and spoke spontaneously for the first time since the onset of his illness, soundly denouncing the scientist. On the way home he spoke more freely to the brother, but upon arriving there relapsed into silence again.

There was no improvement, and on January 23, 1919, the patient came to the Vanderbilt Clinic and on February 6th was referred to us for treatment.

During the first interview, which lasted about one hour, an effort was made to get the patient to speak freely about his difficulties, but while he was willing enough to converse about various topics outside of his illness, whenever this subject was broached he replied that he knew nothing, that he remembered nothing from the time he left his old place of employment until the beginning of his illness. He insisted that he was not getting better, that he was practically blind, and said he had little hope of recovery.

He was assured that the physical examination showed no organic cause for his condition, that the illness was probably due to some difficulty he experienced in adjusting himself to the new job and that a better insight into this difficulty ought to help him to get well. He had no objection to being visited by the social worker who

he was told was to look into the situation more fully and thus help the physician to understand the case better.

Feb. 8, 1919: Social worker visited patient's home and obtained family history and a more detailed account of the onset of the difficulty. The patient accompanied her to his former place of employment at the box factory. He talked freely with the other workmen and showed her where he used to work and how he had to fix the gauges. Walked back with her and talked freely all the way, but on reaching his home he sat down and became absolutely mute, and seemed to be more rigid than before. The wife stated that patient had been better during the past week, assisting with errands and scolding the children when they misbehaved. The procedure of taking him to his old place of employment suggested itself as a possible means of re-attaching him to reality. He had tried two weeks previously to do some simple work at this place but had to give it up.

During this interview the patient seemed to be a little more communicative and much more interested. The additional history of the case made possible the formulation of a theory of the illness. The patient who was quite happy in his old situation, evidently did not relish the idea of going to work for his cousin, but gave in on account of his wife's insistence. He feared that he could not meet the requirements of the new job and wondered why the cousin paid him such a good salary when he didn't think he earned it. As a matter of fact, he did unusually well at his work and even took on duties which were not originally stipulated. But he evidently continued dissatisfied with himself. He found himself in conflict between a desire to return to tasks with which he was familiar, and where he knew he was successful, and a desire to gratify his wife's ambitions and incidentally escape her criticisms. He apparently found the solution in his inability to see the fine work of placing the crystals on the watches, finally insisting that he was going blind, and reacting to criticisms and im-

precations of his wife by an entire withdrawal from reality (mutism and immobility). This theory of the case was gone over with him and although he laughed rather ironically at the idea that he was blind because he wished to be so, he was very much interested. While insisting that he could not see, he wrote from dictation: "I tell the doctor that I cannot see, and yet I am able to write what he dictates." He laughed heartily at this accomplishment and began to talk more freely about himself.

Appears to be very much worried about his relative impotency. Is very much hampered in his sex relations with his wife for fear of causing another pregnancy and they are absolutely determined not to have any more children. Has been practicing premature withdrawal, which both find very unsatisfactory. Patient was told that he might learn of other less disturbing counterceptive measures. It was also disclosed at this interview that home hygiene was not of the best and that some rearrangement would be needed.

Feb. 20, 1919: Social worker reports that the children frequently disturbed the patient at night, but that he objected very strenuously to a rearrangement which would deprive them of the \$5 per month which the cousin pays for his room rent. They would not listen to the suggestion that the rich cousin be appealed to for help, and seemed to be very sensitive on this point.

During to-day's visit the patient talked much more freely about himself, but still insisted that amnesia was no better. He also complained of not sleeping well nights and promised to carry out the suggestion that he occupy the spare room, even though it necessitated the temporary dispossession of the cousin. He admitted for the first time, however, that he was getting better. The wife reports that he is very much better and showing more interest in the home.

Feb. 28, 1919: Patient had not called at the clinic for a week and investigation by

social worker discloses the fact that he has been back at work at the box manufacturing establishment during this period. He is not working at the saws but is helping to handle lumber. The wife stated that he was looking much better, talking and working around the house in a much more normal manner. He has been occupying the spare room, has tried the hot baths before retiring and a glass of beer with evening meal, but is not sleeping much better.

Mar. 8, 1919: For the second time patient has come to the clinic by himself. Appears to have improved materially but still suffers from insomnia. Has been working steadily and has been fairly normal in his relations with his environment.

Mar. 19, 1919: Social worker called at the home and found patient's wife suffering with a heavy cold, a bad headache, swollen eyes, etc. She was naturally feeling blue and depressed, and it was impossible to say how much of her discouraged attitude regarding her husband's health was due to her own physical condition and how much to his actual condition. She stated that he is not much better, is not sleeping at all nights and complains of bad eyes. He cannot see to read and as a result closes his eyes at home. He talks to her quite freely and sometimes plays with the children, brings up the coal and wood, etc., and then goes to bed. Any recreation for them outside of the home is impossible as they cannot leave the children. He is discouraged about his work and is very anxious to get back to his own trade. He is finding the heavy labor difficult owing to his lack of sleep.

Mar. 20, 1919: Social worker called at the Star Box Company and spoke to the foreman regarding patient. Patient is doing fairly well at his work and is receiving the same pay as when employed in the other department. The foreman does not consider that he is physically capable of handling the saws at present as it would be too dangerous for him, but is holding out encouragement that as soon as he is better he can go back to his former work. Says pa-

tient talks freely with other men and acts almost normally, is a trifle rigid, however, and slow in movement of his hands. Foreman promised to encourage him as much as possible.

Apr. 4, 1919: Social worker called at patient's home in the evening and found both man and wife at home. He was playing with the children in quite a natural manner, helping them to mend broken toys. Stated, however, that he is not sleeping more than two or three hours a night, and that he suffers at times from an acute sense of depression and unhappiness which he cannot explain. "Does not feel as he used to feel; feels somehow different." He has gone back to his old job in the saw room but suffers now from a fear of cutting himself with the saws.

He was urged to come to the clinic for further treatment. Tried to explain to him the value of studying the causes of his mental trouble so that he would get completely cured. He has no faith in anything, however, excepting medicine, and feels that he has too much to do to return to the clinic. Half consented to come some Saturday afternoon, if he had the time.

Here, too, it would be premature to claim a complete recovery for the patient and indeed it would be truly striking if such were the case, considering the few treatments we gave him. Neither am I very much inclined to attribute the very marked improvement in his condition and his social-economic recovery to anything approaching a psychoanalytic procedure. It was merely an attempt at giving him a psychological understanding of his difficulty. Such as it was, however, it was very time-consuming indeed.

It strikes me that a clearer differentiation of the members of our staff into those particularly interested in organic issues and those whose special concern are functional disorders, both in the original examination and in the subsequent treatment, would be helpful. Furthermore, a rearrangement whereby part of the staff would work at the clinic forenoons, part in the afternoons

and some selected few at one or two evening clinics, would carry with it greater and better service to a larger number of patients.

We finally come to what we still consider a most important question, namely, the need for a frank recognition of our therapeutic limitations, and how important it is for us to utilize to the fullest possible extent effective social service. It is not absolutely clear to me yet to what extent this social service might be developed as an integral part of the clinic's activities. Some concrete indications have come to light in connection with the cases cited above.

The properly trained social worker could make herself very valuable as an aid in getting a reliable and helpful picture of the setting from which the patients come and to which they have to return to live. Such an estimate of the social environmental situation ought to be of inestimable aid in arriving at a proper understanding of the case, and as a guide in the readjustment of the patient.

The social worker could, furthermore, be very useful in carrying over into the lives of our patients such principles of mental hygiene and correct living as suggest themselves in connection with a given case. She might be of inestimable value in bringing the patient into contact with the available community resources for work, play, and means for a healthy self-expression which so frequently constitute an essential element in the treatment of the maladjustments accompanying disorders of personality.

Finally, she might be utilized as an efficient aid in the after-care of our patients. We have purposely selected in this paper cases which demonstrate some of the therapeutic possibilities in connection with our clinic patients, notwithstanding the limitations and difficulties under which we are compelled to labor, in the hope that it will counteract to some extent, at least, the stultifying attitude of hopelessness which one encounters so frequently in discussing these problems.

AN UNUSUAL FAMILIAL TYPE OF NEURITIC MUSCULAR DYSTROPHY

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The two following cases represent features which are not frequently seen in the neuritic form of muscular dystrophies.

The age of onset and the extent to which the disease has progressed in a short period of time are quite remarkable.

The pathological findings, however, are those commonly observed in the adolescent and adult type of the disease.

Because of the youth of the patients, there are many points of investigation which are necessarily inaccessible, but the findings are sufficiently definite to make these two cases worthy of presentation.

CASE I

Patient, S. R., age five and a half years; female; white. Vanderbilt Clinic No. 24447. Admitted March 26, 1919.

Present Illness beginning with Complaint.—The mother states that the child has never been able to walk alone or sit erect. For the past five weeks she has had incontinence of urine and feces.

Previous History.—She was born after a twenty-four hour labor without instruments and revived with difficulty. She was said to have weighed nine and a half pounds. She was artificially fed and thrived moderately well. Her dentition was normal. It was noteworthy here that she did not hold her head up until seven months, and did not sit up until thirteen months. She began to make attempts at walking at nineteen months, with the aid of a chair, but has never walked alone at any time. She began to talk at sixteen months, but indistinctly. She has never had any accidents. Her tonsils were removed when she was three years old. She had one convulsion when four months old, the cause of

which is not known. She has never been ill except for the above complaint.

Family History.—The parents are first cousins. There is no known history of similar nervous disorder on either side of the family. Her brother is the patient described below.

VOLUNTARY MOTOR SYSTEM.—Cooperation is poor; although the child understands, she seems unwilling to assist.

Abnormal Attitudes and Deformities.—The fingers are flexed. There is bilateral claw hand, more marked in the left. She also has bilateral equinovarus. She has a marked curve of weakness of the back when placed in a sitting position.

Gait.—She cannot walk alone, but stands alone with the feet in the equinovarus position. With assistance she walks in a shuffling manner on the outer side of the soles.

Coordination.—There is no evidence of ataxia of the upper or lower extremities.

Skilled Acts.—These tests are performed as well as her weakness will permit. Her speech is indistinct but can be understood.

Abnormal Involuntary Movements.—These are absent; no fibrillations seen.

Reflexes:

	Right	Left
<i>Deep</i>		
Biceps	0	0
Triceps	0	0
Radial	0	0
Suprapatellar	0	0
Patellar	0	0
Achilles	0	0
<i>Superficial</i>		
Supra-umbilical	0	0
Suprapubic	?	?
Upper lat. abdom.	?	?
Lower " "	?	?

Muscle Strength.—Cannot be satisfactorily tested, but she cannot rise to a sitting or standing position when recumbent. The power of muscle groups could not be ascertained because of the failure of the child to respond to commands. The weakness is quite marked throughout the body but especially so in the distal portions of the extremities and the trunk. She cannot fully extend the fingers of either hand.

Muscle Status.—There is marked atrophy of the distal portions of the arms, the interossei, hypothenar eminences and legs. The muscles are everywhere soft and flabby. Their tone is greatly diminished. There is hyperextensibility of all the joints. Her feet can easily be placed behind her head.

Electrical Reactions.—There is no change in the normal contraction formulæ. There is no reaction of degeneration present, but there is marked tolerance to faradic stimulation.



FIG. 1.

NERVE STATUS shows no abnormality.

GENERAL SENSORY is normal as far as could be determined.

CRANIAL NERVES which are examined in detail show no abnormality.



FIG. 2.

SYSTEMIC EXAMINATION was entirely normal.

LABORATORY TESTS.—The Wassermann blood serum reaction is negative.

Blood examination shows 85 per cent hemoglobin and normal blood smear.

The urine analysis was normal.

CASE II

J. R., age three and a half years; male. Brother of preceding patient.

Present Illness beginning with Complaint.—The patient was brought to the clinic because he falls backward when he attempts to walk. The mother states that he was able to walk well six months ago.

Previous History.—The patient was born at term after a short labor and weighed 4 pounds. There was no evidence of injury at birth. He began to hold up his head at four months, sat up at nine months, and walked at fifteen months. He cut his first tooth at

one year. He began to talk at two and a half years of age. His tonsils were removed because of frequent colds and earaches when he was two years. Otherwise, there have been no other illnesses.

Family History is described in the previous case.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes.*—When his hands and fingers are extended, the proximal phalanges are ex-



FIG. 3.

tended, but the distal phalanges are slightly flexed. Otherwise there is nothing worthy of note.

Gait.—The boy is unable to walk alone and upon making the attempt falls backward. When assisted, his gait is of the equinovarus type with a tendency to shuffling.

Coordination.—Standing with feet together and eyes open, he falls backward without swaying; the same is repeated when the eyes are closed.

Non-equilibratory.—Because of poor cooperation, the tests are not well performed and it is questionable as to the amount of ataxia, if any, in the upper or lower extremities.

Skilled Acts.—There is apparently no abnormality. Speech is of a lisping character but is clear enough to be understood.

Abnormal In voluntary Movements.—None can be detected; fibrillations absent.

Reflexes:

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Biceps	0	0
Triceps	?	?
Radial	?	?
Suprapatellar	0	0
Patellar	?	?
Achilles	0	0
<i>Superficial</i>		
Supra-umbilical	0	0
Suprapubic	0	0
Upper lat. abdom.	1	1
Lower " "	1	1
Cremasteric	2	2
Plantar	1	1
Babinski and modifications	0	0

Muscle Strength.—Because of the child's poor cooperation, it is impossible to state how much diminution there is in the various muscle groups, but there are undoubtedly defects in the distal portions of the upper and lower extremities.

Muscle Status.—The muscles are generally atrophic and of a flabby consistency.

Abnormal Associated Movements.—None.

Electrical reactions were similar to above case.

NERVE STATUS.—Nothing abnormal was noted.

GENERAL SENSORY.—It was impossible to make accurate sensory determinations because of the patient's inability to cooperate.

CRANIAL NERVES.—These were carefully tested and no abnormality was detected.

MENTAL STATUS.—He is bright, active and inquisitive, and is quite normal for a boy of his age.

CRANIAL MORPHOLOGY is apparently normal. No x-ray of the skull was taken.

SYSTEMIC EXAMINATION was normal throughout.

LABORATORY TESTS: The blood Wassermann was negative.

The urine analysis was normal.

The blood smear was normal, hemoglobin 80 per cent.

SUMMARY

The essential pathological features of the above cases may be summarized as follows:

1. The development of marked atrophy of hand muscles before the age of 5 years.
2. Marked disturbance in gait.
3. Absence of all reflexes.
4. Extensive atrophy in the distal extremities.

While it is impossible as yet to eliminate the existence of other myopathic processes, the clinical findings conform more nearly to the neuritic type of muscular dystrophy.

NORMAL AND ABNORMAL VARIATIONS IN THE PITUITARY FOSSA

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That the sella turcica normally varies in size is well known; and as the measurements of the normal variations given by different investigators do not agree very closely, it was determined to study more accurately the normal differences. In order to do this a series of forty normal sellæ were collected. Reproductions of their outlines traced from the x-ray plates are given below. (Fig. 1.) It can readily be seen by glancing at these tracings that marked variations occur in all the dimensions of the pituitary fossa.

The sella turcica is a U-shaped depression in the superior surface of the sphenoid bone. Its anterior, inferior and posterior surfaces are bony, while above and laterally it is covered by projections of dura mater. At its anterior superior margin is the olivary eminence, or tuberculum sellæ, a rounded projecting ridge which is situated below the lower border of the optic groove in which the optic chiasm is lodged. Its anterior and inferior surfaces are formed by a plate of bone which covers the sphenoidal sinuses and is continuous posteriorly with a small bony plate which projects upwards into the cranial cavity and limits the pituitary fossa posteriorly. This projecting plate of bone is the dorsum sellæ. At the superior angles of the dorsum sellæ are little tubercles which are called the posterior clinoid processes. The anterior clinoid processes are the projecting extremities of the lesser wings of the sphenoid, and have not the corresponding relation to the pituitary fossa as the posterior clinoids. The anterior clinoid processes are not anterior to the fossa, but are in a more lateral plane, often being 5 to 7 millimeters external to the lateral margins of the sella turcica.

The anterior clinoid processes serve as

points of attachment for the anterior extremities of the tentorium.

On both sides of the body of the sphenoid, external and inferior to the sella turcica, is the carotid groove which lodges the cavernous sinus and internal carotid artery. The middle clinoid process is situated in this groove just external and below the lateral terminations of the olivary process. Occasionally the middle clinoids are connected by a small spicule of bone to the anterior clinoid processes, and rarely the anterior and posterior clinoids are connected by a bony bridge. (Fig. 2.)

The sella turcica is limited above by the diaphragma sellæ, which is a circular fold of dura firmly attached anteriorly to the olivary eminence, and posteriorly to the posterior clinoid processes. In its center there is a small opening—the foramen diaphragmatis sellæ—through which the infundibulum passes to communicate with the third ventricle of the brain.

The lateral boundaries are formed by a fold of dura which separates the hypophysis from the cavernous sinus.

It is important to note that the antero-posterior diameter is not the interclinoid distance, as is usually given in the literature, but is the distance from the olivary eminence to the posterior clinoid process.

The size of the sella turcica varies enormously, as may be seen from the measurements of the normal given by various authors. The measurements given by Cushing, Gibson, Keith and Fitzgerald are as follows:

	Antero-posterior diameter	Breadth	Depth
	15 mm. (maximum normal)	13 mm.	10 mm. (maximum normal)
Cushing	12 mm.	13 mm.	6 mm.
Gibson	10-12 mm.	14-15 mm.	8 mm.
Keith	10-14.5 mm.	14-17.5 mm.	6.5-7.5 mm.
Fitzgerald			



FIG. 1. TRACED OUTLINES OF 40 NORMAL PITUITARY FOSSA SHOWING THE MARKED VARIATIONS IN SIZE AND SHAPE.

In my series, the measurements of the smallest normal were, anteroposterior diameter, 6 mm., depth, 5 mm.; and of the largest normal, anteroposterior diameter, 15 mm., depth, 12 mm.

In taking radiographs of the sella turcica, it is very important to have the tube and head in the proper position so that there will be no distortion of the outline, and so that the outlines of the symmetrical

on the x-ray plate over the olivary eminence and appear to be the anterior boundary of the pituitary fossa. This is well shown in Fig. 3, where the sella is well outlined, though the anterior clinoid processes are superimposed over the olivary eminence. It is often very difficult to make out the shadow of the olivary eminence, as it is obliterated by that of its anterior clinoids, which are composed of dense bone



FIG. 2. AVERAGE FORMATION OF SELLA TURCICA SHOWING THE POSITION OF THE ANTERIOR CLINOID PROCESSES IN THEIR NORMAL RELATION TO THE PITUITARY FOSSA.

parts will be exactly superimposed. In order to accomplish this the median plane of the skull must be exactly parallel to the x-ray plate. The tube must be directly over the sella, and this corresponds to a point midway between the external angular process of the frontal bone and the external auditory meatus.

In the interpretation of x-ray plates, it is important to remember that the anterior clinoid processes ordinarily do not have any direct relationship to the sella, as they are situated laterally to it, but are projected

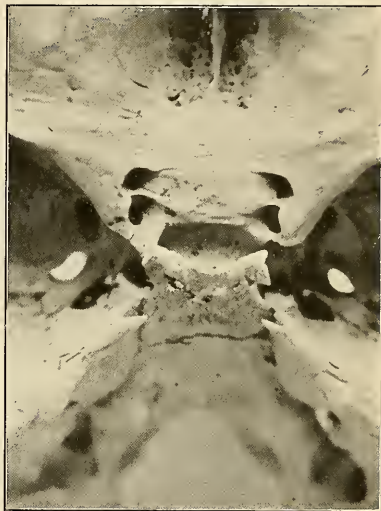


FIG. 3. SELLA TURCICA WITH ANTERIOR, MIDDLE AND POSTERIOR CLINOID PROCESSES ON THE LEFT, AND THE ANTERIOR AND MIDDLE CLINOIDS ON THE RIGHT, CONNECTED BY BONY SPICULES. A FORMATION OF INFREQUENT OCCURRENCE.

and are projected in their longitudinal axes.

The optic groove, or sulcus chiasmicus, is above, anterior and nearly at right angles to the anterior wall of the sella turcica, and this may frequently be made out when it is impossible to see the outline of the tuberculum sellæ.

Figs. 5 and 6 show the outline of the sella and adjoining structures. In Fig. 5 the anterior clinoid processes are projected as though they were within the sella, while in Fig. 6 they appear to be above it.

Usually they are shown in a position between these two, and if they are very long their projection shadow may overlap that of the posterior clinoids and appear to be connected with them. A bony connection

are usually in apposition with the floor of the sella and its anterior wall as high as the lower margin of the tuberculum. The bone forming this portion of the sella is about 1 millimeter thick and is slightly thicker than

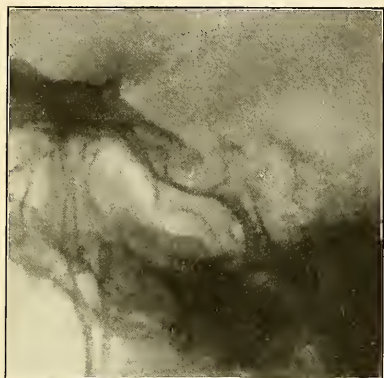


FIG. 4. NORMAL PITUITARY FOSSA.



FIG. 5. NORMAL PITUITARY FOSSA.

of this kind does occur in rare instances, as is shown in Fig. 2; but this is quite uncommon, and when we see this appearance in the radiograph it is usually a projection artifact.

Figs. 3 and 4 show the relation of the

that of the dorsum sellæ or olivary process. The dorsum sellæ is an irregular quadrilateral plate of bone, 5 to 10 millimeters in height, about 2 to 3 millimeters thick, and about 15 to 22 millimeters in width, being broader at the base than at its upper

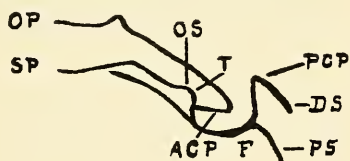


FIG. 6.

- A C P Anterior clinoid process.
- D S Dorsum sellæ.
- F Floor of sella.
- O P Orbital plane.
- O S Optic sulcus.

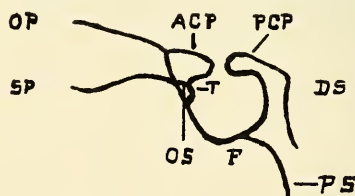


FIG. 7.

- P C P Posterior clinoid process.
- P S Posterior wall of sphenoidal sinus.
- S P Sphenoidal plane.
- T Tuberculum sellæ or olivary process.

Outlines of sellæ in Figures 4 and 5. Note the positions of the anterior clinoid processes as they are projected on the plate; also the tuberculum sellæ which form the true anterior superior boundary of the pituitary fossæ.

floor of the sella to the sphenoidal sinuses. Though the formation of these cells varies greatly, as has been shown by Gibson, they

margin. It is composed of a thin, compact, cortical shell filled with soft spongy bone.

PATHOLOGICAL ALTERATIONS IN THE SELLA TURCICA

Alterations in the sella may take place under the following conditions:

- I. Tumors of the hypophysis.
- II. Tumors of the base of the brain in the region of the pituitary fossa.
- III. Brain tumors and intracranial dis-

trophy. In acromegaly any one of the three following conditions may be found:

1. No change in the sellar size or outline.
2. General enlargement of the pituitary fossa but without any erosion or absorption of bone.
3. Enlargement of the sellar space by absorption of all or of portions of the bony envelope.

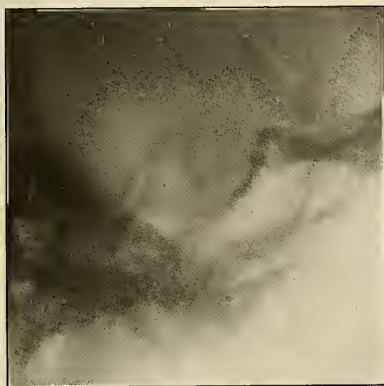


FIG. 8. ENLARGEMENT OF SELLA TURCICA IN ACROMEGALY WITHOUT ABSORPTION OF BONE.

eases producing an increased intracranial pressure.

- IV. Tumors and inflammatory diseases of the body of the sphenoid bone.

I. *Tumors of the Hypophysis.* It is generally conceded now that in all cases of acromegaly there is present either an adenoma or adenocarcinoma of the anterior lobe of the pituitary gland, and with it a glandular hyperfunction. Most of these tumors are adenomata and are of very slow growth, so that there may not be very great enlargement of the gland. Other varieties of tumors which have been described include sarcomata, gliomata, teratomata, chondroma, fibroma, as well as tuberculomas and gummas. If the entire lobe of the gland is destroyed by a hemorrhage, tumor, inflammatory process, or by pressure of a hydrocephalus, the symptoms produced are those of hypophyseal dys-

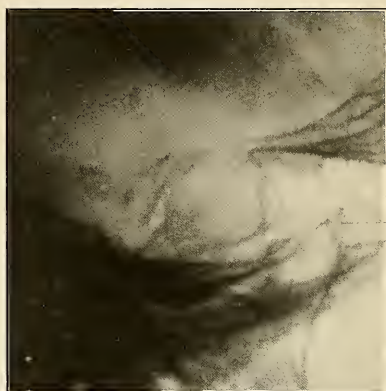


FIG. 9. OUTLINE OF A PITUITARY FOSSA IN ACROMEGALY SHOWING ENLARGEMENT OF SELLA WITH PARTIAL ABSORPTION OF ITS WALLS, THE DORSUM SELLÆ BEING PRACTICALLY DETACHED BELOW WHILE THE POSTCLIVOID PROCESSES REMAIN INTACT.

1. The normal hypophysis, according to Giordani, measures 5 to 8 millimeters anteroposteriorly, 5 to 7 millimeters vertically, and 12 to 15 millimeters transversely. From these measurements it will be seen that the normal hypophysis about fills the pituitary fossa transversely but does not do so anteroposteriorly. This excess space is taken up by the circular sinus, which is composed of two venous channels, passing in front and behind the hypophysis and connecting the cavernous sinuses. Therefore there can be some enlargement of the pituitary gland before it fills its bony cavity.

2. Cases where there is a concentric sellar enlargement. In some chronic cases the pituitary fossa enlarges symmetrically as do the skull and other portions of the bony

framework. This is usually found in the giant type of acromegaly. The sellar measurements of two such giants given by Keith are as follows:

	Anteroposterior	Transverse	Depth
1st case (Giant O'Brien)	21 mm.	24 mm.	11 mm.
2d case	21 mm.	23 mm.	11 mm.

In two other cases where there was slight sellar enlargement he gives the following measurements: 19 mm. x 12 mm. x 8 mm. and 15 mm. x 18 mm. x 6 mm.

3. Fossa enlarged by absorption of bone. This is a very frequent finding in acromegaly. Measurements of such fossæ are given frequently in the literature of acromegaly. Two cases examined by Keith had the following dimensions:

Anteroposterior	Transverse	Depth
20 mm.	17 mm.	20 mm.
19 mm.	22 mm.	14 mm.

As the hypophysis is situated within the pituitary fossa it is evident that in enlargement absorption of bone would not at first involve the tip of the posterior clinoid processes or the top of the dorsum sellæ, but would involve the floor of the sella or its anterior or posterior wall. If x-ray pictures are taken at this stage one may see the posterior clinoid processes and the ventral plate of the dorsum sellæ quite plainly though it may be detached from what remaining fragments of the sellar floor can be made out. This condition is well shown in Fig. 8. Frequently, however, the absorption of the bony space is so extensive that it is impossible to find any landmarks or see any remnants of its outline.

In hypophyseal dystrophy the sella may be normal or enlarged depending upon the cause of the glandular atrophy.

II. In tumors of the base of the brain in the pituitary region the sequence of x-ray findings is quite different from that of intrasellar tumors. In this condition there is first absorption of the posterior clinoid processes and dorsum sellæ, while the floor and anterior wall of the pituitary fossa are at first entirely unaffected.

III. Supratentorial tumors and intracranial conditions, such as chronic hydro-

cephalus, which produce long continued increased intracranial pressure, may bring about absorption of the posterior clinoid processes and dorsum sellæ by causing a protrusion of the adjacent structures into the sellar space. A case of this kind is reported by Cushing. In its discussion he states that "a widening of the cup with more or less absorption of the dorsum sellæ often accompanies marked states of increased intracranial pressure, and is particularly common in cerebellar lesions which have caused an obstructive ventricular hydrops."

IV. Tumors and inflammatory conditions of the body of the sphenoid are uncommon. Metastatic carcinomata and tuberculosis have been described, but are of rare occurrence.

SUMMARY

1. The pituitary fossa is normally subject to great variations in size and outline and gives no information concerning the condition of the pituitary gland unless there is very marked sellar enlargement or absorption of some portion of the bony envelope.
2. The anterior clinoid processes which seem to form the anterior portion of the sella on the x-ray plate, in reality have no close relation to the pituitary fossa, this appearance being an artifact of projection, the true anterior superior boundary being the tuberculum sellæ.
3. Tumors arising in the pituitary fossa cause absorption of the floor and walls of the fossa before affecting the posterior clinoids. Supratentorial tumors and conditions causing chronic increased intracranial pressure may bring about absorption of the posterior clinoid processes.

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A CASE OF BLEPHAROPTOSIS OF PSYCHOGENIC ORIGIN*

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At a recent conference held at the Presbyterian Hospital, Dr. McKendree presented an interesting series of cases of blepharoptosis whose origin could be traced definitely to some underlying pathologic process. As a fitting sequence to his presentation, the case herein reported is of sufficient interest to warrant a detailed description for the following reasons:

1. A thorough clinical study failed to reveal any organic defect to account for the ptosis.

2. The long duration of the symptom—seven years.

3. No improvement of the condition in spite of a varying therapy consistently applied.

4. The definite establishment of an underlying psychogenic defect which unfortunately, however, we were unable to utilize to our advantage, as a psychoanalysis failed to unfathom the mysterious conduct on the part of the eyelids.

HISTORY.—The patient is a female, twenty years of age, born in Russia of Hebrew parentage, who comes under observation for a ptosis of the right lid which is of seven years' duration. Its onset she traces definitely to an inflammatory attack limited to the right eye, which produced redness, swelling and closure of the lid. She visited an eye clinic where a diagnosis of conjunctivitis was made and treatment instituted. After several weeks of treatment the acute phase of the condition subsided except the ptosis which remained. She continued treatment for two or three months and was then referred to another institution. A blood test performed at the first clinic was found to be positive, according to the statement of the patient. At the second institution she came under the care of Dr. Reese, who deemed it ad-

visable to have a Wassermann performed. The result of this test was not communicated to the patient. Two injections of salvarsan were administered without improvement in the ptosis. She continued under Dr. Reese's observation for about two years, during which time several operative procedures were undertaken without result. She was then referred to a prominent neurologist who advised alcoholic injections. She received two of these injections, but on account of the severe reaction she refused further treatment. The ptosis continued, so the patient decided to discontinue all treatment. She thus absented herself for a period of two years. During this time there was no change in the condition. At the end of this time she visited a neurologic clinic where a course of exercises, baths and massage was instituted without avail. She discontinued treatment for another year, during which time there was no change in the ocular condition. She was again seen by Dr. Reese, who kindly referred the case to me.

Leaving aside the ocular condition for the moment we will take into consideration her general behavior during these seven years. She suffered no acute illness but had periods of depression, during which she would isolate herself in her room and refuse to see anyone. At other times, irritability of temper manifested itself and she was unable to agree with those about her. She adopted a negativistic attitude and assumed a sulky, spiteful manner, which conduct brought her into continual conflict with her relatives. They however (the relatives) were quite indulgent and tried to reason with the patient and assure her that their efforts were directed toward helping her in every way, such as advising her to visit

* Presented at a clinical conference June 5, 1912.

the doctor regularly and to take good care of herself generally. She was fed liberally, and was given all she asked for in the way of clothes and money. At one time she was even sent to a private sanitarium in the mountains in order to assist her to a speedy recovery. Her history prior to the onset of the ptosis is suggestive enough to warrant a detailed narrative. That it has a distinct bearing on the development of the ptosis is obvious enough. But the mechanism that brought it about we were unable to determine, as heretofore stated.

She was born in Russia and came to this

for recent and remote events. She was taken to an institution and after remaining there a week was discharged apparently much improved in her mental state. She was then taken to the country and after a stay of three months returned home without slight improvement in the depression. This depression has been persistent up to the present time; but there have been intervening periods in which she has felt quite normal. During these intervals she visits the theatres, calls on her friends and interests herself in all matters of the household.



FIG. 1. BLEPHAROPTOSIS OF PSYCHOGENIC ORIGIN.



FIG. 2. RAISING OF PTOSID LID SHOWING FUNCTIONAL INTEGRITY OF OCULOMOTOR APPARATUS.

country when she was two years of age. Her childhood was without incident. When she was nine years of age, her step-father died and the mother, in order to sustain the family, had to seek employment. Hard work, worry and increasing difficulty to support her children finally undermined her mother's health, and she died when our patient was twelve years old. The death of the mother affected her greatly. She isolated herself, refused to see anyone, cried a great deal and was profoundly depressed. The depression finally culminated in a psychotic outbreak which was characterized by clouding of consciousness, visual and auditory hallucinations, and amnesia

She attended school in a fairly regular manner and managed to finish two years at a high school. Although urged by her relatives to continue, she decided to leave school and seek employment. Her deportment and work at school were up to the standard and elicited no criticism from her teachers.

EXAMINATION

During the examination the patient cooperated willingly and readily answered all questions. She is of average intelligence, keenly analytical as to the variations in her psychic behavior, and evidences a sincere

willingness to assist in any way that will expedite her recovery.

VOLUNTARY MOTOR SYSTEM.—There were no abnormal attitudes of the voluntary motor system noted except the ptosis, which will be considered more in detail under the examination of the cranial nerves.

Gait.—This is normal in all its phases.

Coordination.—All coordinative movements, both of equilibratory and non-equilibratory type, are performed in a normal manner.

Skilled Acts.—All skilled test acts showed no defects and were satisfactorily performed. There was no dysmetria noted. All rapidly repeated pronation and supination movements were well executed.

Reflexes.—The deep reflexes of the upper and lower extremities were present and equal and showed no variations from the normal.

Abnormal Involuntary Movements.—There were no demonstrable tremors, twitchings, athetoses, choreiform movements or spasms.

Muscle Strength.—This shows no impairment in the entire somatic musculature. All possible forms of muscular effort, such as flexion, extension, rotation, abduction and adduction, were performed in a satisfactory manner.

Muscle Status.—There was no atrophy or hypertrophy noted. Hypotonia and hypertonia likewise absent.

Abnormal Associated Movements.—There were none demonstrable.

NERVE STATUS.—There were no impairment in the behavior and no pathologic alterations noted in the condition of the peripheral nervous system.

GENERAL SENSORY.—General sensory examination revealed acuity, localization and discrimination normal for touch, pain, temperature pressure and muscle-tendon sense.

CRANIAL NERVES.—As the ptosis was the paramount disturbance noted thus far, the condition of the oculomotor apparatus was examined with particular care. The pupils responded promptly to light and accommodation. The consensual reaction was normal. There was no limitation in the ocular movements of the right eye after raising of the ptosed lid. When the supporting finger was removed the lid would promptly snap into place. When the patient was requested to raise it voluntarily, one could readily discern a marked spasmodic tremor in the upper and lower lids and a slit-like aperture was the result of the effort, through which the sclera of the eye was barely visible. Conjugate eye movements were normally performed. The fundi were negative. The other cranial nerves were carefully investigated and found functionally intact.

SYSTEMIC EXAMINATION.—The patient is much undersized and shows very poor bodily development. Menstruation has been but recently established. These factors would indicate a marked dysfunction in the endocrine system. There are no other noteworthy abnormalities noted.

LABORATORY EXAMINATION.—All laboratory data have been uniformly negative.

SUMMARY AND DIAGNOSIS

What are the deductions to be drawn in an analysis of this case? A very thorough clinical examination of the cerebrospinal axis reveals no organic abnormalities. There are no gross motor or sensory disturbances. The movements of the right eyeball in all planes are performed readily enough. But the ptosis remains persistently as mute evidence of the only neurologic abnormality. We can hardly consider this of organic origin and ignore the escape of the other ocular muscles from a similar fate. Laying aside the organic theory as

untenable we obviously have to consider the ptosis as a functional affair. Perhaps the term psychogenic as applied to this particular case may be inappropriate. Shall we name it a reflex orbicular spasm? The adherents of the reflex theory will readily find abundant testimony to substantiate their belief, for we have not a history of an eye infection and a subsequent develop-

ment of the ptosis. Reflex irritability readily enough. But that hardly disposes of the psychogenic idea. The underlying psychic defect is there. The history is clear enough on that point. The soil was ripe for the development of this bizarre paralysis. The eye infection was simply the release that culminated in this abnormal functional behavior.

CONGENITAL AMYOTROPHIC ATAXIA

BY JOHN H. NOLAN, M.D.

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CASE

J. H., male, American, age fifteen years. Case History 24360 V. C. Admitted to the Vanderbilt Clinic March 17, 1919.

HISTORY. Present Illness.—His mother states that he is not able to walk any distance alone because he staggers and falls. His father thinks he is improving in his gait.

Previous History.—He is the second child of healthy parents, born normally after a two and a half hour labor; he cried at once and presented no evidence of injuries. He weighed eight pounds. He was breast fed for seven months and then placed on various formulæ. He became very sick with gastro-enteritis and lost several pounds, so that when he was a year old he was very feeble. He could not hold his head up until he was two years old, and began to learn to walk at seven years, but he was not able to walk alone until he was eleven years. He cut his first tooth at one year and could talk at one and a half years. His anterior fontanelles did not close until he was three years.

He is said to have had infantile paralysis at the age of two years because of his wasted and weakened legs, but his mother states that he could not move his legs long before this illness. He had measles at three years, uncomplicated; pneumonia at five years and again at seven years. He has never had any accidents or operations.

Family History.—His parents and one older sister are in good health. A younger sister died of hemorrhage following cutting of the lingual frenum when three days old. There have been two miscarriages, causes unknown. There is no known familial nervous disorder in his family. The parents are not related.

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—He has a marked lordosis when he stands and a curve of weakness when he sits. He has a mild degree of pes cavus.

Gait.—He has a tabetic gait, but also staggers when he walks.

Coordination.—He sways when standing with feet together and eyes open and falls in a few moments unless supported, but with eyes closed, he falls at once. Non-equilibratory test shows ataxia in the right arm and left leg. The left arm and right leg movements are normally performed. He pass-points to the left with the right hand but performs normally with the left.

The tests for dysmetria and adiadochokinesis show that they are present.

Skilled Acts.—Normally performed. Speech is normal.

Abnormal Involuntary Movements.—Fibrillary twitches are observed under both arms and on the back.

Reflexes:

(a) Deep	Right	Left
Jaw	o?	o?
Pectoral	o	o
Biceps	o	o
Triceps	o	o
Radial	o	o
Ulnar	o	o
Wrist	o	o
Suprapatellar	o	o
Patellar	o	o
Achilles	o	o
Dorso-cuboidal	d	d—dorso-flexion
Periosteal	o	o
(b) Superficial		
Cilio-spinal	1	1
Supra-umbilical	o	o
Suprapubic	o	o
Upper lat. abdom.	o	o
Lower lat. abdom.	o	o

Cremasterics	I	I
Plantar flexion	I	I
Babinski	O	O
Chaddock	O	O
Oppenheim	O	O
Gordon	O	O
Schäfer	O	O

Muscle Strength.—He arises with difficulty and with the use of his arms to a sitting position. He also makes use of his arms to a great extent in rising to a standing attitude. His general muscle strength is diminished in all extremities, but particularly so in the right arm and left leg. His grasp is stronger in the left hand than in the right, but is greatly diminished in both.

Muscle Status.—There is wasting of the thenar muscles. His general musculature is poorly developed, especially in his arms and legs. The muscles are soft but the tone is normal. The pectoral muscles exhibit myotatic irritability and myoedema in a slight degree. The electrical tests were not done.

Abnormal Associated Movements are not present.

NERVE STATUS shows no abnormality.

GENERAL SENSORY.—Touch, including localization and discrimination; pain and temperature, normal.

Vibratory sensation is absent over the left foot and half-way up the left leg, elsewhere it is normal.

Muscle tendon sense is greatly diminished, if not actually lost, in the toes of both feet, particularly in the left big toe. There is also diminution at the left ankle and knee.

Stereognosis is normal.

CRANIAL NERVES.—The olfactory, acoustic, trigeminus, facial, glossopharyngeus, spinal accessory and hypoglossus nerves are normal.

The optic, visual apparatus, oculomotor nerves and oculomotor apparatus: The vision is myopic to a slight extent. The

fundi are normal. The pupils are 3 mm. wide; they are equal, regular, central, and react promptly to light and accommodation. There is no strabismus, diplopia or nystagmus. The eyes exhibit considerable exophthalmus.

MENTAL STATUS.—Considering his very late start in school at the age of nine years, he has made exceptional progress.

Systemic.—The tegumentary examination shows no abnormality.

The glandular system seems to be normal.

Cardio-vascular.—The pulse is regular, 72 per minute. The heart is normal, and the systolic blood pressure is 110.

The respiratory, gastro-intestinal and genito-urinary functions are normal.

He has a well marked macroglossus.

Laboratory Tests.—The Wassermann blood serum was negative.

SUMMARY

We are dealing with a case whose symptoms have been present from infancy. He was very ill at seven months, but a baby should be able to hold its head up alone at four months. He was late in learning to talk, sit, stand and walk but he has been gaining more control as he grows older.

The examination shows combined ataxia, loss of equilibration, adiadochokinesia and dysmetria. Fibrillary twitchings are observed in both arms, associated with a noticeable degree of wasting of the thenar eminences. The deep reflexes are absent; and of the superficial, only the ciliospinal, cremasterics and plantar are preserved. His muscle strength is greatly diminished and his muscle status shows poor development.

The sensory examination reveals pallesthesia in the left foot and leg as described above, combined with marked disturbance in the muscle-tendon and joint sense in both feet, left ankle and knee.

Anatomically, we would place the lesion

in the dorsal columns, the columns of Clarke, and the lateral cerebellar tracts. This would explain the disturbance of deep sensibility. There would necessarily be an involvement of the ventral horns to account for the fibrillary twitching and atrophic changes in the muscles.

Clinically, are we dealing with an atypical Friedreich's? His gradual improvement speaks entirely against this syndrome, although I believe it approaches this class more than any other. Tabes can be ruled out by the serological reaction and the normal pupils.

It seems more reasonable to assume that there has been a congenital anomaly present. This would explain his gradual improvement, probably due to the fact that he is using cortico-spinal tracts which are in good condition.

DISCUSSION

DR. OSNATO.—The case suggests to me rather a definite type of Friedreich's ataxia than one of neuritic muscular dystrophy; and putting the case anatomically into its place, we must admit immediately that the ataxia here is the type that one sees in tabes that is not cerebellar in type. The loss of deep muscle sense, tendon sense and vibratory sense would also point to some trouble in the posterior columns. The ataxia, of course, means the spino-cerebellar or lateral columns. The atrophy in the small muscles of the hand would point to the anterior horn cells. He has nystagmoid movements, a scoliosis, and a suggestion of the Friedreich's foot in the big toe. This latter is a very uncertain symptom, as many of the cases never have it until very late. The Friedreich's foot is an indication of pyramidal irritation plus muscle atrophy with connective tissue substitution for muscle and contracture of the same.

Anyone who has seen a number of cases of Friedreich's would immediately refuse to accept the opinion that the anatomical

trouble is limited to the posterior half of the cord. Neither one of the Brown children whom we studied here had much sensory loss. Even as keen an observer as Dr. Strong failed to find any in the boy, yet his deep reflexes were absent. In early cases of Friedreich's, one of the first symptoms to appear is the diminution or loss of deep tendon reflexes. Therefore, the trouble cannot be on the afferent side of the arc, but must be in the anterior horn cells. I have yet to see a case where there was not some atrophy in the thenar and hypothenar muscles of the hand and in the lower extremities. I cannot explain the atrophy in any way except on the basis of involvement of the anterior horns.

DR. ARONSON.—I would suggest that a skiagraphic examination be made of this boy's sella turcica, as there are signs that justify a suspicion of pituitary disease. Pituitary hypoglycemia as an etiological factor in the dystrophies is admitted. I am inclined to think that the patient looks dystrophic; he has a very sad and aged face. His sadness is pathological for his youth. He has considerable pigmentation and hypertrichosis. His genitals are three times the size they should be. This does not explain his agenesis. I have found diminished vibratory sensibility in the left lower extremity and absence of the same from the middle of the tibia down, but aside from this, one cannot help thinking that the boy has also dyspituitarism or some analogous endocrinological state.

DR. WECHSLER.—The case does not seem to fit in with what we are accustomed to look upon as Friedreich's ataxia. That disease usually begins at about seven or eight years of age and grows progressively worse, while Dr. Nolan's case apparently began at birth and has shown gradual improvement. It is true that there are some symptoms which are found in Friedreich's, but they can be explained by the anatomical involvement. The nystagmus is doubtful in this case and so is the pes cavus; besides, the latter is only a secondary muscular result and not an index of

what has taken place in the cord. It seems to me that a congenital agenesis of the columns of Goll and Burdach and the spino-cerebellars would better explain the case under consideration and also account for the Friedreich symptoms. Against the latter there is also the absence of pyramidal tract signs. However, one need not quarrel too violently over a pathological label. The whole condition points to a congenital

agenesia with progressive improvement rather than a developmental abiotrophy with progressive aggravation.

DR. OSNATO.—May I make a suggestion in the way of therapeutics? We had some advanced cases of Friedreich's ataxia at the Kings County Hospital who have improved markedly with reeducation. You can teach these cases to walk just as you can teach a tabetic by reeducation.

ACUTE HYDROCEPHALUS

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Acute hydrocephalus is a general term and no more specific than pleural or pericardial effusion. Serous meningitis is used synonymously in the literature, but should imply an effusion of inflammatory nature. Most of the reported cases are of inflammatory origin as these are the most common and the most important, a meningeal transudate usually being part of a terminal process in a chronic illness such as nephritis, diabetes, or cardiac disease, and its symptoms not easily separated from those produced by the general disorganization of the vital processes.

A serous effusion in the cranial cavity may be due to irritation of a mechanical, chemical, thermal or infectious nature. The excessive accumulation of fluid may be within the ventricles, producing an internal hydrocephalus; in the subarachnoid space, causing an external hydrocephalus, or a general distention of the ventricles and subarachnoid spaces of the brain and cord.

The process may be further localized, and instead of being a diffuse ependymitis may be localized to one lateral ventricle by an occlusion of the foramen of Monro, or to the fourth ventricle by obstruction of the aqueduct of Sylvius. There may also be occlusion of the foramina of Key and Retzius; but obstruction of these foramina is not necessary for the production of acute hydrocephalus, and probably does not occur in the majority of cases. The most frequent cause is some disturbance of the normal balance of secretion and absorption of the cerebrospinal fluid. On the exterior of the brain and cord localized serous effusion may be formed, giving rise to "pseudo tumors." Various grades of inflammatory changes have been described in the ependyma, choroid plexus, meninges and brain substance. The character of these

pathologic changes depends largely upon the inflammatory agent and may vary from simply a slight thickening and edema of the affected tissues to more pronounced changes of an ependymitis or leptomeningitis.

When the effusion is produced by mechanical, chemical or thermal irritants, the symptoms are frequently serious and quickly follow the causative injury. They are often fatal and do not respond to treatment as well as those of infectious origin. The cases of infectious origin are the most interesting and the most important. The symptoms in this form may come on gradually and the source of the infection is frequently obscure. The differential diagnosis is usually difficult, as in the subacute cases brain abscess or tumor is closely simulated. The very acute cases may be confused with sinus thrombosis or purulent meningitis.

In the reported cases of this last variety, the most frequent point of entrance of the infection is through the ear. Whether organisms of attenuated virulence gain entrance to the subarachnoid space, or whether the effusion is caused simply by an absorption of toxins from an epidural abscess, has given rise to some discussion. Some observers believe this latter explanation to be the most plausible, and in support of it call attention to association of pleural effusion with sub-diaphragmatic abscess.

A subarachnoid serous effusion may be present in some infectious diseases, such as pneumonia, typhoid fever, scarlet fever, influenza, measles and acute articular rheumatism, and give symptoms of meningeal irritation—the so-called meningism. While it is possible that meningism and serous meningitis may be due simply to the toxins which circulate in the blood and produce

an aseptic ependymitis, it seems more likely that they are both due to infections of low virulence. Whether the condition be meningism, serous or purulent meningitis, is probably a question of the virulence of the infecting organism. In the case reported below the condition must have been due to an infection of the ependyma by an attenuated organism, and not to any toxic substance, because the primary focus of infection had entirely cleared up before any signs of hydrocephalus appeared.

The symptoms of acute hydrocephalus may be general or local, but depend principally on the increase of intracranial pressure. The general symptoms are headache, vomiting, weakness, listlessness, somnolence or stupor, convulsions, delirium, pain in the neck, abdomen or extremities, and choked disc. The focal symptoms which have been noted are paralysis of the cranial nerves, hemiplegia, hemianesthesia and aphasia. The spinal fluid usually shows evidence of a subacute inflammatory process in an increased number of cells which as a rule are lymphocytes, though at times polymorphonuclear leucocytes are present also. It is clear and colorless and usually under greatly increased pressure. Globulin is present in varying amounts and sugar is usually absent. Cultures are generally sterile.

The course of the disease is usually irregular. The acute cases may end in recovery or death. The recovery may be complete or leave some permanent paralysis or optic atrophy. In the subacute and chronic cases the course is usually not steadily progressive, but is marked by temporary improvement and exacerbations. Lumbar puncture or decompression produce rapid improvement.

The treatment consists in relief of the intracranial pressure through lumbar puncture, decompression or puncture of the corpus callosum. Quincke advised the administration of mercury, and salvarsan has been given in some instances, but as a great many cases recover spontaneously it is difficult to judge of its usefulness, and in

the absence of any sign of syphilis it is probably superfluous.

CASE

PATIENT: J. C., aged seven years; school girl; American.

HISTORY. *Chief Complaints*.—1. Double otitis media five weeks ago; duration two weeks. Then a free interval of five or six days.

2. Paroxysmal frontal headache
3. Vomiting
4. Listlessness
5. Loss of appetite
6. Constipation
7. Drowsiness.

Duration, two weeks. Course, progressive.

Family History.—Both parents are living and well. Mother had four stillbirths. Dr. Cragin made the last delivery and is reported as having said that the fetus had died in the seventh or eighth month and that the cause of death had been similar to that causing fetal death in syphilis; but in this case there was no evidence of syphilitic infection. The mother had two blood Wassermann tests made at the Sloane Hospital and both were negative. There is no tuberculosis in the family and no known exposure to tuberculous persons.

Personal History.—She is a first child; birth was normal, feeding and development were normal. She was always an active child; lived in a private home in good surroundings, having plenty of fresh air. The appetite, bowels and urination were normal until the onset of the present illness. She attends a public school and is in Grade 3 A.

Previous Illnesses.—Adenoids removed two years ago. She had measles one year ago. For the past two years she has occasionally talked in her sleep and walked during her sleep a few times. No fever or night sweats. She has gained in weight steadily. Two months ago she weighed 56 pounds. She has had no colds or respiratory infections during the past winter.

Present Illness.—The onset was sudden five weeks ago with discharge from the

left ear. There was no pain or fever. A physician was called who ordered irrigations. One week later the right ear became painful and the ear drum was incised. Both ears were irrigated for the next week. Then the discharge lessened and the patient was pronounced cured. An interval of a week followed during which the child was perfectly well and appeared normal. Two weeks ago the present symptoms began. Headache, which is paroxysmal and definitely localized, has been the chief complaint. It began in a small area immediately above the left eye, and later (five weeks ago) it began in a similar area above the right eye also. The pains begin suddenly, are very sharp and piercing for a few minutes, becoming more intense, and then gradually lessen. They do not radiate. The duration of each attack is from five to eight minutes. The intervals between attacks vary widely from fifteen to thirty minutes during the day to several hours during the night. From the onset the child has appeared tired after each attack. For the past five days the pain has been more severe and more generalized. During the past week the child has been very listless—for three days really drowsy, taking several naps during each day. Her sinuses were examined a week ago and said to be normal.

The vomiting is not severe, though she is frequently nauseated. She has vomited five times in all and this within the past two weeks. It is not projectile. She has complained of nausea more than pain.

The appetite was good until five days ago; then she refused all food and has taken nothing but water since.

The bowels have been regulated with medicine or enemata; otherwise they would not have moved. The poor appetite could in part account for the constipation.

Drowsiness has been progressive. The lassitude after attacks of headache became continuous listlessness, and later drowsiness.

There have been no injuries; no recent nasal or respiratory infections; no exposure

to contagious disease; no fever or dizziness, diminution of vision or convulsions.

PHYSICAL EXAMINATION

General Appearance.—Well nourished, well developed little girl of seven years; she walked into the ward and complained of headache over the eyes. She is rather pale and appears acutely ill.

Head is normal in size with a rather high forehead. There is no tenderness over the sinuses or other portions of the skull. *Eyes:* Pupils are equal, regular, react readily to light and on accommodation. Vision is normal. Examination shows a bilateral papilledema which is more marked on the right. *Ears:* Ear drums appear normal. *Nose* is normal; no discharge. *Mouth:* Breath is foul; tongue is coated and dry; teeth are dirty. Pharynx shows no signs of inflammation. There is absolutely no rigidity of the *neck*. When the neck is fully flexed the patient says that it causes pain over the eyes.

Thorax is thin; expansion is good and equal on the two sides. There is no irregularity of respiration; respiratory rate is 22.

Lungs are clear.

Heart is normal in size and position; pulse rate is 52, it is slightly irregular. There are no thrills or murmurs. The sounds are of good quality at both base and apex.

Abdomen is somewhat scaphoid-shaped. The muscles are held rather rigidly. There appears to be tenderness in the left iliac fossa. *Liver:* Dullness extends $1\frac{1}{2}$ inches below the costal margin; its edge is not felt. *Spleen* is not palpable or enlarged to percussion. There are no abnormal masses or signs of abdominal fluid.

External genitalia are normal; there is no vaginal discharge.

Extremities are normal. There is no Kernig's sign, no twitching or automatic movements.

Superficial Lymph Nodes: No pathological enlargement.

Temperature: 98. *Weight:* 48 lbs. 14 oz.

NEUROLOGICAL EXAMINATION, 3/3/18

VOLUNTARY MOTOR SYSTEM. *Attitudes*.—

There is no spinal curvature, abnormal attitude or deformity of any of the extremities.

Gait.—The gait walking forward slowly or quickly is perfectly normal; it is also normal when walking with the eyes closed. There is no abnormality brought out by Fournier's test.

Coordination.—There is no swaying on standing with the feet together either with the eyes open or closed. There is slight swaying in all directions if she stands on either foot, but no difference with the eyes open or closed. Non-equilibratory tests in both upper and lower extremities are normal. There is no dysmetria or adiadochokinesis.

Skilled Acts.—There is no dyspraxia, the patient being able to feed herself or salute with either hand. Speech is normal. There is no dysarthria or paraphasia. The handwriting is normal for a child of seven.

Reflexes:

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Jaw	1	1
Pectoral	1	1
Biceps	2	2
Triceps	1	1
Radial	1	1
Ulnar	1	1
Wrist	1	1
Suprapatellar	Absent	Absent
Patellar	1	1 (elicited only on reinforcement)
Achilles	Absent	Absent
<i>Superficial</i>		
Ciliospinal	Present	Present
Supra-umbilical	Present	Present
Suprapubic	Present	Present
Upper lateral abdominal	Present	Present
Lower lateral abdominal	Present	Present
Plantar flexion	Present	Present
Babinski	Absent	Absent

(1 = slight; 2 = active; 3 = very active)

Abnormal Involuntary Movements.—

There are no tremors, twitchings, choreiform movements, spasms or convulsions.

Muscle Strength.—Normal throughout and equal on the two sides.

Muscle Status.—Contour is normal; consistency and tone are normal. There is no abnormal irritability.

Abnormal Associated Movements.—None present.

NERVE STATUS. *Irritability*.—Trousseau's sign absent. Chvostek's sign absent.

GENERAL SENSORY. *Touch*.—Acuity, localization and discrimination normal. Pain normal. Muscle tendon sense and stereognostic perception normal.

CRANIAL NERVES AND HEAD. *Olfactory and Nose*.—There is no nasal abnormality. Smell is normal on each side.

Optic and Oculomotor Apparatus.—Vision: Right eye 20/40; left eye 20/40. Perimetry: normal. Eye grounds: Marked choked disc on each side. Pupils regular, centrally placed; position of mid-dilation; they react actively to light and on accommodation. There is no hippus. The palpebral fissures are normal and equal on each side. There is no exophthalmos or enophthalmos; no strabismus or other ocular deviation. Conjugated eye movements are normal and there is no nystagmus.

Acoustic Nerve and Ear.—Hearing is normal on each side. Air conduction is greater than bone conduction.

Trigeminus Nerve and Mouth.—The movement of the jaw in all directions is normal. Corneal reflex is present on each side. There is no anesthesia.

Facial Nerve and Face.—Normal on each side.

Glossopharyngeus and Vagus, Pharynx and Larynx.—The uvula is situated in the mid-line. Palatal and pharyngeal reflexes are normal. There is no difficulty in swallowing. Phonation and the quality of the voice are normal.

Spinal Accessory Nerve.—There is no weakness of either sternomastoid or trapezius muscle.

Hypoglossus and Tongue.—The tongue is protruded in the mid-line and its movements in all directions are normal.

SUMMARY OF NEUROLOGICAL EXAMINATION.—Double choked disc with diminution of visual acuity in each eye. Knee jerks diminished—elicited only on reinforcement. Achilles jerks absent.

LABORATORY EXAMINATIONS

2/11/18	Throat culture: No Klebs-Loeffler bacilli	
	Cervical smear: Few pus cells; no gonococci	
	Blood count:	
	White blood cells	16,800
	Polymorphonuclear leucocytes	80%
	Lymphocytes	18%
	Mononuclears	2%
	X-ray of Skull:	
	Sella turcica well defined	
	Clinoidal process well shown	
	No definite indication of increased intracranial pressure	
	X-ray of Lungs:	
	No indication of any pulmonary disease	
2/12/18	Lumbar puncture. Spinal fluid:	
	Amount	18 c.c. [colorless
	Appearance	Perfectly clear and
	Pressure	Apparently normal
	Cell count	52
	Lymphocytes	97%
	Polymorphonuclear	
	leucocytes	3%
	Globulin	+
	Wassermann	Negative in all dilutions
	Guinea-pig inoculation:	Autopsy did not show any sign of tuberculosis
2/12/18	Von Pirquet reaction negative	
2/13/18	Blood Wassermann:	
	Alcohol antigen negative	
	Cholesterin antigen +	
2/19/18	Lumbar puncture. Spinal Fluid:	
	Amount	12 c.c.
	Pressure	Normal
	Appearance	Perfectly clear
	Cell count	Lymphocytes 67
	Globulin	+
	Tubercle bacilli	None found
2/23/18	Blood count:	
	White blood cells	14,200
	Polymorphonuclear leucocytes	75%
	Lymphocytes	25%
2/25/18	Von Pirquet reaction negative	
2/25/18	Urine Analysis:	
	Specific Gravity	1.026
	Reaction	Acid
	Color	Amber
	Turbidity	None
	Precipitate	None
	Albumin	None
	Glucose	None
	Microscopical examination	Negative

CLINICAL NOTES

2/13/18. After the lumbar puncture the patient seems clinically much improved. She has had little or no headache, but cried during the night with pain in the abdomen.

2/20/18. A few days after the first lumbar puncture the headaches seemed severe again and the patient became more drowsy and listless. Yesterday she had a second lumbar puncture and since then she is much improved. She complains of no headache, has had a good appetite and is bright. She spends most of the day talking

with the other children. The improvement in her mental condition is very striking.

2/23/18. The patient again complained of a good deal of abdominal pain this morning. She was given an enema which resulted in the removal of considerable constipated feces. As the pain still continued a few hours later, she was given a colon irrigation which removed a considerable amount of brown, soft fecal material. After this the abdominal pain ceased and it is quite probable that all the abdominal pain has been the result of constipation only.

3/5/18. During the patient's stay in the hospital (from February 11th to March 5th) she has improved until at present she feels perfectly well. The improvement has been marked after each lumbar puncture. There has been no other treatment except rest and occasional catharsis. The eye grounds still show a moderate swelling of each disc; but she no longer complains of headache and is not dizzy or listless.

3/29/18. The patient was again admitted to the hospital with the following interval history. She left the hospital three weeks ago greatly improved, with no headache, nausea, vomiting or other trouble. She says that she felt perfectly well until March 23rd when she developed severe abdominal pain and also a severe headache. She also felt very dizzy and her headache was made worse by walking or any exertion. She consulted her physician who advised her to return to the hospital. Her mouth is very dry. She has no appetite and is unable to eat anything without being nauseated.

Physical Examination at this time gave almost the same findings as the examination on admission. It is entirely negative except for a choked disc on each side, slight rigidity of the neck, slight epigastric tenderness and diminished knee jerks. The temperature is 99; pulse 84; respirations 24; blood pressure 85 = 55; weight 48½ lbs.

4/1/18. X-ray of sinuses showed no indication of sinus disease.

4/13/18. The blood of the mother in Wassermann reaction showed:

Alcohol antigen	negative
Cholesterin antigen	negative

4/19/18. *Lumbar puncture. Spinal fluid*, removed under increased pressure, perfectly clear. Examination of the fluid not made.

4/20/18. Five-tenths gm. diarsenobenzol was given intravenously with no reaction.

4/23/18. *Ophthalmoscopic Examination* (by Dr. Schoenberg).—Left eye: The margins of the disc cannot be made out. The veins are dilated. Several white spots are seen in the region around the disc in the retina. The choked disc measures 5 diopters. Right eye: The condition is similar to that seen in the left; the choked disc measures 6 diopters. There are no signs of optic atrophy. Vision in the right eye is 20/40 +; in the left eye 20/40 +.

4/25/18. Following lumbar puncture the child's symptoms cleared up at once. However, headache and drowsiness with nausea returned in two weeks, which were immediately relieved by a second lumbar puncture. The patient was seen by Dr. Tilney, who considers the diagnosis acute internal hydrocephalus. It has been suggested that she be given a course of anti-specific treatment. One injection of salvarsan was given with no reaction. Feeling perfectly well, she is allowed to go home.

4/30/18. *Ophthalmoscopic Examination* (by Dr. Schoenberg) showed papilledema 4 diopters in the right eye; $4\frac{1}{2}$ diopters in the left. Vision 20/40 in each eye.

5/1/18. Patient was given 0.4 gram salvarsan intravenously with no reaction.

Blood Wassermann: Alcohol and cholesterin antigens negative.

5/7/18. Since the last lumbar puncture there have been no headaches, vomiting or drowsiness. Her eyesight has been normal. She is able to read without glasses. She was given 0.4 gram salvarsan intravenously today.

5/14/18. The patient returned to the hospital for another salvarsan injection and states that she has been in very good condition since last admission, with no vomiting or headaches, and that she has returned to school. Four-tenths of a gram of salvarsan was given intravenously.

Blood Wassermann: Alcohol and cholesterin antigens negative.

Ophthalmoscopic Examination showed the right disc 4, the left disc $4\frac{1}{2}$ diopters.

Lumbar Puncture. Spinal Fluid: 25 c.c. of clear spinal fluid removed under markedly increased pressure at first, but at the end under practically normal pressure, simply dropping from the lumbar puncture needle.

Appearance	Clear, colorless			
Cells	19 lymphocytes			
Globulin	+			
Culture	Negative			
Smear	No tubercle bacilli			
Wassermann	2 c.c.	1 c.c.	0.6 c.c.	0.2 c.c.
Alcohol	Negative	Negative	Negative	Negative
Cholesterin	Negative	Negative	Negative	Negative

7/25/18. The patient has been entirely free from symptoms since the last note.

Ophthalmoscopic Examination (by Dr. Schoenberg) shows in the right eye a choked disc of 2 diopters; in the left eye a choked disc of 3 diopters.

8/26/18. The patient was admitted to the hospital for lumbar puncture and ophthalmoscopic examination, but stated that she had been entirely free from symptoms for the past four months.

Fundus Examination (by Dr. Schoenberg) showed both eye grounds entirely normal, the choked discs having completely disappeared. Visual acuity was normal. Both visual fields were normal.

<i>Lumbar Puncture. Spinal Fluid:</i>				
Pressure	Normal			
Appearance	Perfectly clear and colorless			
Cell count	8 lymphocytes			
Globulin	Negative			
Wassermann	2 c.c.	1 c.c.	0.6 c.c.	0.2 c.c.
Alcohol	Negative	Negative	Negative	Negative
Cholesterin	Negative	Negative	Negative	Negative

11/26/18. The patient was last seen on this date when she was perfectly well, having had no symptoms for the past seven months. The eye grounds were normal.

SUMMARY

The points of interest in this case are:

1. The onset of general symptoms of increased intracranial pressure after an attack of double otitis media.
2. The striking relief of symptoms after lumbar puncture.
3. The occurrence of marked choked discs with visual disturbances, both of which eventually entirely disappeared.

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THE INSIGNIFICANCE OF LOCAL DISTURBANCES IN THE PRODUCTION OF CONSTITUTIONAL NERVOUS SYMPTOMS

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The above caption is not adopted with the purpose of discrediting the earnest investigation that has been carried on, especially in recent years, in the field of the psychoneuroses, as well as in organic disorders of the nervous system. It is well known that because of better diagnostic methods the number of cases that come at an early period under the neurologist's observation is decidedly on the increase. It is well known also that the therapeutic armamentarium of the neurologist is greatly circumscribed; and I believe that nowhere in the entire field of medicine is there greater need for experimental, scientific, carefully interpreted investigation of constitutional nervous symptoms. Therefore the work is to be encouraged. But in the course of the past ten years particularly, the enthusiasm of many sincere efforts has led to widespread haphazard, unscientific therapy on the part of the profession. It is because of this fact that I have undertaken a study of the psychoneuroses, with especial reference to local causative factors. I have arrived at certain definite conclusions, which in my opinion should serve as a check upon the present trend of management of these cases.

During the past two years it has been my privilege, in the department of therapy, to treat and observe thirty-five hundred cases of organic as well as functional disease. I

have been alert to the possibility of focal infection, reflex irritation, and visceral disease in the production of the nervous phenomena, and with the assistance of the various departments of the Vanderbilt Clinic careful records of their findings and interpretation have been filed.

By the expression "constitutional nervous symptoms," I refer to those complaints usually made by the so-called neurotic individual. They include headache of long duration, chronic states of insomnia, irritability, generalized pains of a vague type; easy fatigue, compulsions, fears, maladjustments, depression, "indigestion" and so forth.

These patients demand relief. They are incapacitated from carrying on their occupations, and their families suffer. The situation commands interest on the part of the physician, and realizing, with a sense of helplessness, the inadequacy of the old-time therapy he is more than ready to adopt new suggestions. For years he neglected to perfect his knowledge of psychotherapy, and it was only after remarkable enlightenment by the sects which dealt only with the psyche of an individual, or by suggestive methods which reached the psyche indirectly, that he awoke to the new possibilities. But he meets with failure even with psychotherapy. Perhaps even constructive psychoanalysis has failed. And

consequently he turns to the newer ideas in an effort to reach the patient, and to accomplish a maximum amount of relief. The patient will submit to any procedure if he is promised relief; hence the temptation is great to find a localized process which may account for the generalized symptoms. The patient to-day who is suffering more or less chronically, goes the rounds, eyes, sinuses, tonsils, appendix, pelvic organs, colon, and if his symptoms are those of the constitutional type, he sooner or later returns to the neurologist with fundamentally the same complaint. I do not wish to be misunderstood in my position regarding the importance of correcting local disturbances if the patient's health permits. But I do wish to emphasize what I believe to be paramount, namely, the importance of taking into consideration the individual's lack of nervous stability, the strain under which he is laboring, anxiety which may surround his whole life, and his disregard of proper hygienic laws. I maintain that local pathological processes may serve as coincidental or even exciting factors in producing the psychoneurotic manifestations; but the physician who allows his enthusiasm to promise a cure by giving attention only to the local process is bound to be sadly disappointed.

The chronic sufferer begs for a definite tangible reason for his illness. And if his physician with great reassurance states that at last the source of trouble has been found, already the patient experiences relief. The demand for a definite tangible diagnosis was forcibly brought to my attention by a recent experience. A well-known practitioner called me in consultation in a case of cerebral thrombosis. The patient presented a most interesting mixed aphasia. Together we spent an hour and a half with the problem, largely with the purpose of accurately localizing the lesion if possible. Much to my surprise, the following day my colleague was notified by the family that he need not call any more, as they had found a physician in the neighborhood who knew just what the trouble was. The new

physician may not have even recognized the aphasic features, but even reassurance arising from total ignorance would have been acceptable to the family. It is just so in dealing with neurotic individuals. The field is most fertile for the exploitation of new ideas, and everything from refractive errors to chronic appendices are gladly accepted by the suffering patient as an explanation of his distressing symptoms.

But I must add a word of caution which can be borne in mind by all of us to advantage. Frequently organic systemic disease is masked by hysterical or other neurotic manifestations. One of my most troublesome neurotic patients who had made her home almost intolerable by excessive irritability, selfishness in the extreme, by frequently insulting members of her family and by devitalizing forty nurses, and twelve to fifteen doctors, was found to have carcinoma that had invaded practically every bone in her body. And previous to having had x-ray assistance, the clinical picture resembled a border-line psychosis very closely. Such mistakes in diagnosis have led clinicians to make extensive and thorough examinations of all neurotic cases. And it is due to such examinations that pathological local disturbances are discovered, and errors of interpretation which I am about to elaborate are committed.

The following represent some of the chief complaints made by neurotic patients, to which I have given special study. And with reference to some of the symptoms I shall refer to the local lesions to which they are often attributed.

Headaches of long duration at once invite our attention chiefly to refractive errors, sinus involvement, nasal obstruction, toxemia, neoplasm, syphilis, and arteriosclerosis, or allied disease. Of course I assume that the last four factors have been ruled out by careful analysis. In my experience, refractive errors lead to a definite type of headache, which bears a direct relation to the use of the eyes. In chronic headache, I have never known simple cor-

rection of the error entirely to relieve the patient. Moreover, in neurotics, time and time again I have known four to six skillful oculists to vary decidedly in their interpretation of the amount and kind of error. By all means should refractive errors be corrected as nearly as possible, but no opinion is trustworthy that has not been based upon a careful history of the patient's daily life, physical and mental.

Sinusitis also is a most important consideration in the production of headache. Especially so, if one obtains a history of recent infection or a series of infections. But here again it has been my experience to find a certain type of pain, variable in extent and intensity, with certain clear-cut indications that the sinuses are involved. The finding of a simple catarrhal exudate, without reference to recent infection, does not satisfy me in the interpretation of headache.

Gross nasal obstruction with blocking of the sinuses demands immediate attention, but I have not known spurs, moderately deflected septa, or simple enlarged turbinates to give rise to headache as a chronic complaint.

Toxemia, especially of the intestinal variety, has not quite finished its day as a haven of refuge for the baffled neurologist. The laboratory report which includes a finding of four plus indican and a toxic stool immediately suggests definite treatment, and a way out for the consultant. But the patient has his course of irrigations, and his remarkably variable diet, and returns still complaining of headache. A further course of treatment is considered necessary, but unavailing, the patient is sent away to the country, where his daily life is ordered, his responsibilities and anxieties reduced to a minimum, and he progressively improves. Are we always justified in comforting ourselves by believing that the patient could not have responded so satisfactorily if it had not been for our previous careful watching of his indicanuria?

Convulsions, of the so-called idiopathic

type, represent another variety of disturbance with which the medical man is constantly struggling. Focal irritation is always hoped for, but with vascular disease, neoplasm, syphilis, or fracture completely ruled out, it is never found. Toxemia in adults has for years furnished us with an avenue of hope, but as an explanation has been of small service to the patient. Infection has certainly enjoyed its short life as a causative factor. In babies, toxemia is of great importance, and should be eradicated at once, but convulsions appearing in infancy should always be regarded as an indication of a neuropathic tendency. Dentition, in my opinion, plays an insignificant rôle in the production of convulsions. Preputial and clitoridean adhesions are relegated to the same position.

Chorea I mention only as an indication of nervous instability. It is undoubtedly a systemic disease, the result of infection. Its close association with rheumatism leads me to speak of tonsils and adenoids as an avenue of infection. I have known hundreds of recurrences after tonsillectomy and adenectomy have been done, although in several cases I have seen prompt subsidence of the choreic manifestations soon after operation.

Indigestion as a prominent symptom requires first investigation for ulcer, malignancy, cholecystitis, appendicitis, adhesions, etc. In the patient presenting constitutional nervous symptoms, can we confidently expect the eradication of a gastric ulcer to relieve all the symptoms? Do adhesions cause psychoneuroses? Does a chronic appendix affect an individual's nervous stamina? How often do we read of the close association between hyperchlorhydria and gastric ulcer! How often is hyperchlorhydria dependent entirely upon a neurotic tendency! The surgeon finds adhesions, and is surprised that they are not nearly so extensive, or multiple, as they are in many other individuals upon whom he has had occasion to operate for some other urgent indication who had no symptoms of indigestion. He is forced to conclude that in

neurotic individuals, hypersensitiveness gives rise to symptoms which in normal persons are unnoticed. Chronic appendicitis with its train of distressing complications, demands immediate attention from the surgeon, but in at least fifty cases which I have carefully studied, removal of a diseased appendix, with its coincident adhesions, has not removed constitutional nervous symptoms.

The visceroptoses, discovered in the course of an examination of a psychoneurotic, are of course regarded as functional disturbances. Increase of fat deposit, abdominal support, abdominal massage, moderate exercise, and rest after meals often produce remarkable results. I believe that increasing the nervous tone by the above procedures is responsible for the improvement. And I firmly believe that the visceroptoses are the result of an impoverished nervous tone, rather than the cause.

Mucous colitis is met with very frequently in our daily practice. I think that most gastroenterologists admit the very intimate relationship between this distressing malady and the neuroses. Even in babies, when it is found not to be due to a definite dietetic error, the nervous system must be given careful attention. While I have known considerable improvement to result, I have never found the general nervous manifestations to yield entirely, unless the local treatment has been combined with measures directed toward improving the general nervous and physical tone of the patient.

Pus foci are still flourishing, in the general medical field as well as in the neuropsychiatric realm. Apical abscesses and unhealthy tonsils, because of resulting chronic absorption, have given rise to the most bizarre explanations of many hitherto inexplicable disorders. And I do not wish to belittle their importance in certain instances. But with reference to the production of constitutional nervous manifestations, I wish to be clearly understood that my experience does not justify their importance. Time and time again the dental

surgeon and the throat specialists have reported their positive findings, with the assurance that absorption was most certainly taking place. Acting with full confidence that interference was to the best interest of the patient, I have had these abnormalities corrected. Undoubtedly the patient's best interest was served in the matter, but I reiterate that the nervous symptoms were not substantially relieved. I will state in brief the results obtained in five cases.

CASE I

C. B., a girl of seventeen years, had suffered for ten years with daily headache. The pain became so severe at times that she had to give up her school career. She has been unable to work steadily at her trade. She was repressed, shy, and unsocial. Careful examination by several clinicians failed to reveal any organic abnormality. Refractive errors were corrected. The blood picture was negative. Neurologically nothing could be determined. The only point of attack lay in buried, unhealthy tonsils which a conservative throat expert considered worthy of removal. Tonsillectomy was accordingly done, and to-day, four or five months later, the patient's complaint remains identically the same.

CASE II

R. I., thirty-six years of age, has had headaches daily for fifteen years. She has been otherwise typically neurotic. Six months ago she was advised to have her tonsils removed, as they were buried and decidedly unhealthy. Today she suffers from the same type of headache, although she admits it is somewhat lighter in intensity. Incidentally she has been much freer from responsibility, has been careful with her diet, and has had more open air life.

CASE III

X. Z., a man of sixty-four years of age, became markedly depressed, with delusions of persecution and self-depreciation. It was

discovered in the course of a thorough investigation that several teeth had infected apices. Accordingly several teeth were removed by a dental surgeon. Intestinal toxemia was also pronounced and colon irrigations were instituted at once. The patient steadily progressed downward over a period of three months, and finally ended his life. I might add that he had had several similar attacks of depression previously, and was in my judgment, a typical manic-depressive.

CASE IV

A. B., a Russian Hebrew, of middle age, complained of a bitter, salty taste in her mouth, a burning sensation in her tongue and lips, headache, pains all over her body, insomnia, and a desire to die. She was negative throughout except for several infected apices and an area of necrotic bone in the mandible. These were removed at once. Six weeks after the operation she had experienced no relief from her distressing symptoms.

CASE V

H., an aviator, of superb physique, was discharged from the army because of irresponsibility. He had previously been normal in every respect. He became exalted, verbose, sleepless, and extravagant. Brought under the care of an eminent internist, apical abscesses were found and immediately removed. A few weeks later he barely escaped imprisonment for passing worthless checks. He has been committed to a sanitarium where he has exhibited all the signs of a manic-depressive individual.

To summarize, then, my experience not only in these cases but in hundreds of others, in my judgment *pus foci* do not have a direct bearing upon the production of chronic headache, of psychoses represented chiefly by the cyclothymias, or in the franker types of psychoneuroses.

Pelvic displacements and pelvic disease in women have for a long time furnished material for investigation along the lines suggested above. But I am sure that the gynecologist is now regarding them with much more conservatism. I am sure that he finds retroversion, for example, in many healthy, rugged women, who are unconscious of any defect. I am sure that he notes at once the temperamental difference between the scrubwoman who goes about her daily work happy though prolapsed, and the dragged out, headachy, neurasthenic individual who may have backache as the symptom of a simple retroversion. He recognizes that fixation and rest in bed for two or three weeks, followed by a rest in the country, produce remarkable results. But I am confident that the majority of conservative gynecologists do not fail to place the credit for the relief of general symptoms where it belongs.

In a sense the above may seem to consist chiefly of destructive criticism. But I have endeavored only to put a check upon our enthusiasm, which has led us quite apart from fundamental therapy. With the advent of psychoanalysis, certain well-selected cases may be cured without recourse to other measures. The endocrinologists are coming forward with the promise of real assistance in solving the perplexing problem of therapy. But in all cases attention to the detailed physical and mental life of the patient is absolutely essential. Food, rest, exercise, fun, and congenial occupation must be given careful consideration in planning the patient's régime. All disturbing pathological processes must be rationally corrected if possible. We cannot disregard them. But we must impress ourselves as well as our patients with the old-time paramount importance of upbuilding the morale and physical tone by rational living combined with stimulating psychotherapy.

JUVENILE PARALYSIS AGITANS*

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INTRODUCTION

True juvenile paralysis agitans has always been looked upon as a very rare if not doubtful entity, but in recent years active attention has been focused on its existence. It is true that a few of the cases reported bore closer identity with so-called pseudosclerosis of Westphal or progressive lenticular degeneration of Wilson, or were merely early, if not transitional, cases of multiple sclerosis; but there is no doubt that some were actual Parkinsonian syndromes occurring in youth. It becomes necessary, therefore, to exclude all other possibilities before one makes bold to proclaim the existence of a true juvenile Parkinson.

The recent epidemic of encephalitis, which existed along with that of influenza, brought forth a number of typical paralysis agitans syndromes and, as the disease more frequently attacked the young, we observed many cases which might have been called juvenile Parkinsons. The term, however, would not be strictly correct. In the first place the cases were acute; secondly, the syndrome was but one part of a general picture, and thirdly, most of them made partial or complete recoveries. Nevertheless the occurrence of those cases served to call attention to the pathologic process which may possibly underlie paralysis agitans and perhaps point the way to an understanding of that disease. As soon as we learned to localize more or less definitely the site of the lesion in paralysis agitans a new light was shed on the various explanations of its pathogenesis. Indeed we began to recognize that for the production of the syndrome, it matters little what the pathologic process be as long as it confines its slow destructive activity to

a definite part of the corpus striatum and to it alone. A gliosis akin to multiple sclerosis, which is not multiple, an arterio-sclerotic focus, an atheromatous patch, a slowly degenerative change either of vascular or non-vascular origin, a slowly progressive syphilitic or neoplastic encroachment,—all may give a true picture of paralysis agitans. Of course many of these postulated causes have little more than theoretical foundation, but none is so far-fetched as to make it impossible; and as some or even all of them may at times affect the young, one may account for the existence of a Parkinson at any age. It is unfortunate that incipient cases are not brought under the microtome to help us in the study of the actual causes.

The case to be reported below is undoubtedly a true paralysis agitans; but whether it is juvenile or not depends on the flexibility of the term juvenile. Parkinsons nearly always occur late in life, and very rarely before the fifth decade. Strictly speaking, a typical, slowly progressive paralysis agitans occurring in the third decade, that is, beginning at twenty-five, cannot be called juvenile, but its rarity at that age and its close approach to the second decade make that designation not altogether unwarranted. And while the patient definitely assigns twenty-five as the date of onset it is not unlikely that the destructive process causing the disease has preceded the clinical manifestation.

In view of the possible doubt attaching to the caption a detailed description of the case becomes necessary. It should be emphasized that our patient's condition had nothing to do with the recent epidemic of influenza. Whatever the cause, the disease antedated the epidemic by two years.

* Presented at a Clinic Conference, Neurological Department, Vanderbilt Clinic.

CASE REPORT

History.—H. L., male, single, age twenty seven and a half years, furrier, was first seen in May, 1919, when he complained of shaking of the body, more particularly the right arm and leg. The tremor began very slowly early in 1917 and had gradually become worse. At first the right arm began to tremble, after several months the right leg followed suit and finally the tremor spread to almost all the limbs. All the extremities, but particularly the right arm, have become weak. Our patient complains of drawing pains in the right arm. He had to give up work on account of the tremor, which is constant by day but disappears at night. The tremor is always present at rest, and according to the patient is intensified by worry or excitement or quick effort. He does not drop things. The whole body has become stiff and all his movements are slow. He has difficulty in walking, says that he runs when he gets up to walk, he complains of pain in the back and finds it difficult to get up or sit down. He lost all sexual desire and does not think he is potent, not having indulged for six months.

The patient's habits are good: he does not drink tea or coffee, does not smoke or drink, although he used to indulge moderately in the former and excessively in the latter, at times even getting drunk; he denies masturbation. His sleep is normal, appetite good, bowels regular. He used to weigh 180 pounds, at present weighs 166. He has no pain or difficulty in passing water, urinates four or five times daily, once at night; but he speaks of urgency in micturition. No incontinence. He has no respiratory difficulties, never had any injury to the head or other parts of the body, no convulsions or other motor disturbances and no operations. He sees well and never saw double. Hearing is normal, no vertigo or headache. He denies venereal diseases and never was sick in his life. His mental and emotional states have not altered. Family history is negative, par-

ents, three brothers and three sisters being alive and healthy, one sister having died in confinement; no history of tuberculosis, epilepsy, insanity, alcoholism or paralysis agitans in ascendants or collaterals. The present condition was not preceded by fever or malaise.

Physical Status.—Voluntary Motor System. Mask-like, immobile face, stiff attitude, stoops forward, arms and hand held rigidly by the sides and a little in front of the body. The gait is stiff, rigid and in one piece. There is marked propulsion and retropulsion. He falls back in one piece in the chair on trying to sit down. Coordination is normal. All the tests are slowly but accurately performed. Skilled acts are somewhat interfered with, mainly by a stiffness and slowness of execution. The handwriting shows a characteristic tremulousness. Speech is slow, dull, but not quite monotonous. Of abnormal involuntary movements, he shows a rapid, rhythmic, fine tremor of the fingers of the right hand, at rest and on action. There is a regular tremor of both lower limbs, moderate in amplitude and medium in rate.

Reflexes:

	Deep	R	L
Jaw.....		1	1
Pectoral.....		2	2
Biceps.....		2	2
Triceps.....		2	2
Radial.....		2	2
Ulnar.....		2	2
Wrist.....		2	2
Suprapatellar.....		?	?
Patellar.....		2	2
Ankle.....		2	2
Dorsocuboidal.....		0	0
Periosteal.....		0	0
	Superficial	R	L
Ciliospinal.....		1	1
Supra-umbilical.....		2	2
Suprapubic.....		2	2
Upper lat. abdom.....		2	2
Lower lat. abdom.....		2	2
Cremasteric.....		2	2
Plantar flexion.....		1	1
Babinski.....		0	0
Chaddock.....		0	0
Oppenheim, etc.....		0	0

All the deep reflexes were difficult to elicit but lively when obtained.

Muscle Strength.—All movements showed

adequate strength but were slow in being initiated. There were no atrophies or hypertrophies. There was rigidity in the muscles of the lower extremities and slight in the upper, the right more than the left. So-called cogwheel movement in the right upper. All associated movements in the face and limbs were abolished.

General Sensory.—Touch, pain, temperature, vibratory, muscle-tendon and stereognosis normal all over.

Cranial Nerves.—Smell normal. Vision 20/100 in each eye; fields and fundi normal. Pupils normal in size, shape and position; react promptly to light and convergence. There is no strabismus. There seems to be a slight limitation of movement of the right external rectus, probably normal. No nystagmus. Palpebral fissures normal, von Graefe is present. Cornea normal. Hearing normal. Motor fifth normal, conjunctival reflex present; teeth and mouth normal. Taste normal; facial movements are probably normal although there appears a doubtful weakness of the right face. The uvula is somewhat deviated to the left, but its movements and all movements of the tongue are normal. The palatal and pharyngeal reflexes are present.

Mental Status.—Intelligence normal, emotion dull, attention and memory good, no phobias, delusions or hallucinations.

Systemic.—Skin and hair normal. Glands not palpable. Pulse 84, heart normal, blood pressure 130 S., 85 D. All else negative. Wassermann negative. Urine showed trace of albumin and a few granular and hyaline casts; sp. gr. 1.026.

CONCLUSION

From the above history and examination and subsequent observations there seems to be no doubt that our patient represents a typical, slowly progressive paralysis agitans. The absence of corneal changes, mental degeneration, speech disturbances, liver changes, etc.; the comparative slowness of progress, and the rather late onset rule out both pseudosclerosis of Westphal and lenticular degeneration of Wilson even if there had been any reason for considering those diseases as possibilities. The only question, therefore, is whether one is justified in tacking on the word juvenile to the diagnosis. Elasticity of the term may be taken as justification. In any event the case is of interest and perhaps not unworthy of record because of the very early date of onset and its great rarity.

As to etiology one can merely offer a suggestion. Certainly an acute inflammatory origin is excluded. The urinary findings and comparatively high blood pressure may point to arteriosclerotic changes, but why they should have picked out only the extrapyramidal system of fibers is not at all clear, although it is not less mysterious than the selective action, if it does occur, of a similar process in advanced age. The youth of the patient need not exclude arteriosclerosis, as we know that it may occur at almost any age and even in children. However, any attempt at explanation is merely speculative and for the present we must content ourselves with a plain record of observation.

THE PATHOLOGICAL CHANGES IN THE CEREBELLUM IN ACUTE ANTERIOR POLIOMYELITIS

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In reviewing the literature of acute anterior poliomyelitis, one finds the pathological alteration in the cord and medulla described in great detail, but little is given of the changes in the cerebellum. The cases showing marked ataxia are described by some authors as "the cerebellar type," and by others as "the ataxic type." Ataxia has been frequently observed and described and may occur with varied lesions in the brain and cord. Peabody, Draper and Dochez⁸ say that ataxia "may well depend on an upper motor neuron lesion, either in the brain or cerebellum, in the pyramidal tracts or in Clarke's columns." Weisenberg^{12, 13} believes that there is no such thing as an ataxic type, as the ataxia may be associated with lesions in many portions of the nervous system, and often the disturbance of gait is simply the result of weakness of the limbs.

Coordination is accomplished, in a broad sense, after the manner of a reflex and requires the integrity of the afferent and efferent nervous pathways. Lesions of the peripheral nerves sensory of the motor cord tracts may cause ataxia as well as lesions of the cerebellum or cerebral cortex. The ataxia type of poliomyelitis, however, should be limited to the cases where the ataxia is the outstanding feature and is not accompanied by paralysis, or at least not limited to the paralyzed portions. In every epidemic of poliomyelitis there are cases described in which ataxia is the principal feature. The child may have gone to bed apparently normal and awaken the next day practically unable to walk. The gait is slow and uncertain, the steps of irregular length with the feet widely separated. The body sways from side to side and the child stumbles and falls. There is

incoordination of the movements of the upper extremities shown by the difficulty in grasping or picking up small objects with the hands. Nystagmus is also present at times. There may also be cranial nerve lesions or muscular palsies.

In cases of this type it seems probable that the main lesions are in the cerebellum, though they might conceivably be located in the coordinating pathways of the mid-brain, or possibly in the spino-cerebellar tracts.

That there are cases in which the lesions are mainly cerebellar is well recognized and a number have been reported in the literature. There is of course some question as to whether the etiological agent is the same as that of acute anterior poliomyelitis, but in some instances it must be, and there are changes in the cerebellum in some cases of typical anterior poliomyelitis.

The ataxia may disappear very rapidly, or it may require months, but complete recovery has taken place in practically all of the reported cases. Two cases in which the recovery was striking are reported by E. S. Taylor¹⁰ and are of sufficient interest to be quoted verbatim.

"M. D., age ten, was admitted from the neurological out-patient department into clinical in June, 1913, for inability to walk. Her parents were both healthy and there was no nervous disease in the family. A brother and two sisters were all healthy. Patient had scarlet fever in July, 1912, but there were no sequelæ. Nine weeks before admission patient's two sisters had measles, and one week later the patient herself complained of sick headache and was kept in bed one week. On getting up again she was noticed to nod her head

about from before backwards, though showing no movement when lying down or when asleep. Four days later she was unable to stand up and complained of a sharp pain in the left chest radiating to the groin. The nodding of the head was quite independent of talking or of any attention paid to her.

"On examination the patient was found to be healthy in appearance and able to lie quite still in bed, but when raised to a sitting posture and asked questions the movements of the head at once commenced. The speech was slow and hesitating but hardly scanning in character. The knee-jerks were present and both plantar reflexes were flexor. The pupils reacted normally and were equal in size, and there was no nystagmus. When placed upon her feet she exhibited well marked 'asynergia,' falling backwards continually and showing extreme ataxia. On the day of admission the patient complained that as she walked the room 'seemed to go round,' but this symptom did not persist.

"On June 6th, two days after admission, the movements were less marked and on June 7th she could stand unassisted, the movements of the head were less and she was able to talk more readily. On June 8th the walking was much improved and on June 12th the patient could walk without assistance. On June 14th the ataxia had almost gone and the patient was discharged eight days later. In this case no drug was given as treatment."

"P. P., age seven, was admitted into clinical on July 11, 1913, for inability to walk. The family history showed no nervous disease and there were six other children alive and healthy. The patient was a full term child and labor was normal. She had measles and chickenpox when a baby. Two days before admission the child walked to school in the morning, a distance of about one mile, and seemed quite well. At midday as she was walking out of school she suddenly fell down and was unable to stand up without assistance. She could not walk alone and was carried

back by her sisters. She did not vomit, but complained of headache the next morning.

"On admission the patient was a well developed child and answered questions rationally, but seemed apathetic. Her speech was not normal, the words being spoken in an abrupt manner and in a monotonous tone. Her temperature was 99.2. Both knee jerks were present; the plantar reflexes were flexor and there was no muscular wasting. There was no optic neuritis, the pupils reacted normally, and there was no nystagmus. When lying in bed the patient made no abnormal movements but on sitting up she moved her head irregularly and could not maintain the sitting posture without assistance. When standing up she was completely ataxic with rocking movements of the head in all directions and irregular movements of the arms and body, and when trying to walk she showed a constant tendency to fall backwards unless supported. She was not giddy and made no effort to support herself when falling. There was intention tremor of both arms. There was evidence of old chronic otitis media in the left ear, but tests carried out showed no other aural defect, the labyrinth being normal on both sides.

"A lumbar puncture was performed and 3 c. c. of cerebrospinal fluid were withdrawn. The fluid was under normal pressure, contained no cells nor organisms and was quite clear. The patient was at once given urotropin in 10 grain doses every six hours. She had an evening temperature varying from 99 to 101. Babinski tests for cerebellar disease were all positive, 'adiadochokinesia' and 'asynergia' being well marked.

"On July 15th the movements were much less and the intention tremor was very slight, though the ataxia remained unchanged. On July 16th the patient could almost stand alone and on July 19th she could walk a few steps alone holding her head to the left. On July 21st the speech was normal and the patient could walk with

her eyes closed. On July 22d 'adiadochokinesia' was still present but the other signs were not definitely given. There was some frequency of micturition and hematuria. The urotropin was therefore discontinued. On July 28th the urine was normal and four days later the patient showed no signs of cerebellar disease, her gait, speech and arm movements being absolutely normal."

Another case of cerebellar polioencephalitis is reported by Frederick Taylor.¹¹ The patient, a boy of four years, developed

mentioned in the literature are briefly as follows:

Wickman¹⁴ simply states that the cerebellum presents changes analogous to those found in the cerebrum. Abramson¹ says:

"The changes in the cerebrum and cerebellum were very mild when compared with those in the cord. The most common change consisted of engorgement of the vessels, the endothelium of which had the appearance of being swollen. In four-

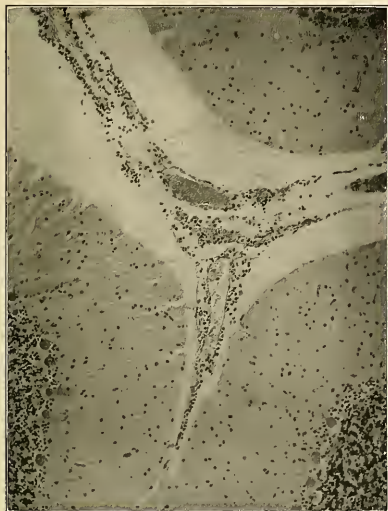


FIG. 1. CEREBELLAR PIA SHOWING INFLAMMATORY CHANGES.

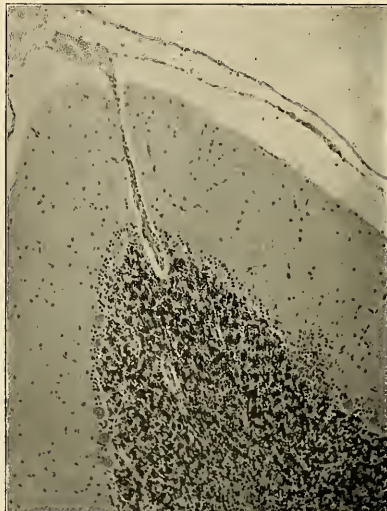


FIG. 2. SLIGHT VIRCHOW-ROBIN SPACE INFILTRATION IN A BRANCH OF A MENINGEAL VESSEL; ALSO A SLIGHT FOCAL CELLULAR ACCUMULATION IN THE MOLECULAR LAYER.

a tremor, ataxia of the cerebellar type and nystagmus, drowsiness and vomiting. The drowsiness was present about a month, the attacks of vomiting a little longer, but the extreme ataxia continued for over two years and it was more than three years before there was complete recovery.

Pathological changes in the cerebellum are absent in many cases of poliomyelitis, are usually not marked, and are often difficult to find when present. The changes

teen of thirty-seven cases the brain exhibited very mild cell reactions, consisting in slight degree of perivascular infiltration, and in a few cases in an accumulation of cells apart from the vessels. The nerve cells exhibited no degenerative changes. The cerebellum exhibited similar changes in five of thirty-three cases examined. These changes were most commonly found below the cellular layers in the cerebrum and below the granular and Purkinje cell

layers in the cerebellum, in among the fibers radiating from the cortex."

Batten² describes the lesion as a thrombosis of the finer terminal arterioles.

Blanton³, in the description of material obtained from fifteen autopsies, says one case microscopically "showed a meningitis over the whole brain including the cerebellum . . . basal ganglia and pons. In none of the twelve cases in which the cerebellum and cerebral hemispheres were studied could one make out

type of infiltration as that of the cord, but was not so extensive nor so uniformly present. In one very early case the infiltration in the pia in the cerebellar region was very intense. . . . The cerebellum showed very little inflammation. The pia of its middle lobe usually showed considerable lymphocytic infiltration and in one case there was a considerable subpial hemorrhage in that locality."

Neal and Abramson,⁷ in their description of the ataxic type of poliomyelitis,

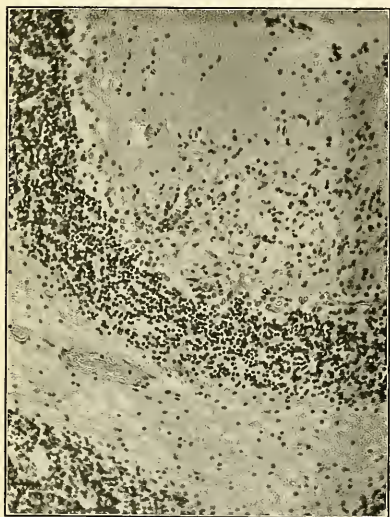


FIG. 3. FOCAL CELLULAR ACCUMULATION AND EDEMA IN THE MOLECULAR LAYER.

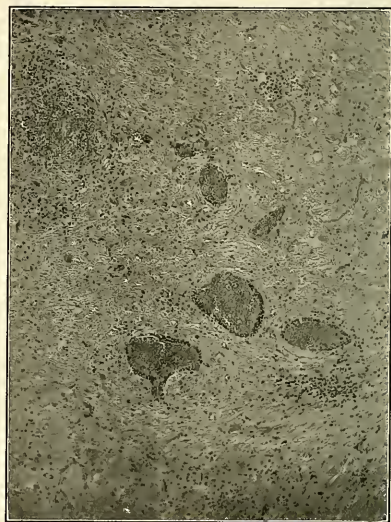


FIG. 4. VASCULAR CONGESTION; VIRCHOW-ROBIN SPACE INFILTRATION; HEMORRHAGE AND FOCAL CELLULAR ACCUMULATION IN THE DEEP WHITE MATTER OF A CEREBBELLAR PEDUNCLE.

any changes save those of congestion."

Cadwalader⁴ found the following in the cerebellum in one case: "The pia is slightly affected and there are a few small hemorrhages in the cortex."

Strauss,⁹ in his article on the pathology of acute poliomyelitis, says: "The pia covering the medulla, pons, crura cerebri, the interpeduncular space, the corpora quadrigemina, and the middle lobe of the cerebellum, in my cases showed the same

state: "The motor nerve cells are evidently not involved, but there is lack of coordination, ataxia, nystagmus, etc. In some cases an ataxic gait is the only sign of involvement of the central nervous system other than the sensory symptoms. The anatomic basis for this is proved by the postmortem findings of involvement of the cerebellum (Clarke's column) and the intervertebral ganglia. This type is very rare."

Harbitz and Scheel,⁵ in the study of their cases, found that the "cerebellum usually presented few evidences of inflammation, and only in the central portions which were connected with the medulla and pons."

Miller,⁶ in a study of a case of acute poliomyelitis, gives the microscopical changes in the cerebellum as follows: "Besides congestion of the vessels, several small patches of cell infiltration are seen especially in the deeper nuclei. One roof nucleus is very severely affected. The folia of the vermis and the lateral lobes have practically escaped; only a very few vessels show slight perivascular cell infiltration. No areas of necrosis nor evidences of reaction are found in the cerebellum."

PATHOLOGICAL FINDINGS

Macroscopic Changes.—Frequently there is nothing to be seen on gross examination of the cerebellum. The surface is moist and shining and at times somewhat congested. The convolutions may be flattened due to the increased intracranial pressure. On section the cerebellum is rather edematous and the general congestion may be noted by the diffuse reddening of the surface.

MICROSCOPIC CHANGES

I. Tissues of Mesodermal Origin.

Pia Mater.—The pia mater frequently shows diffuse inflammatory changes. This is evident not alone on the surface of the convolutions, but is present in the smallest interfolial clefts. This inflammatory reaction is usually much more marked in the cerebellar pia than in that of the cerebral hemispheres where there is found more of a diffuse congestion and edema. The changes in the cerebellar pia are very similar to those seen in the cord. The membrane is swollen and edematous. Its layers are widely separated by the congested blood vessels, exudated cells, edema and hemorrhage. The appearance is markedly different from that seen in

ordinary meningitis not alone in the character of the cells involved but because of the lack of fibrin and absence of any exudate on the pial surface. Hemorrhages are present within the pial meshes but they are usually not large enough to be evident macroscopically. The infiltrating cells are largely mononuclears of the lymphocyte type. There are, however, some large mononuclears, polyblasts, and polymorphonuclear leucocytes.

Blood Vessels.—The blood vessels of the pia and throughout the substance of the cerebellum are dilated and congested. Thrombosis has never been observed and, contrary to the opinion of Batten, probably plays no part in the pathology of anterior poliomyelitis. Hemorrhages are observed in the molecular layer of the cortex and deep in the central white matter, and occasionally are seen infiltrating the area between the molecular and granular layers.

In poliomyelitis the most striking vascular change is the cellular infiltration of the Virchow-Robin or adventitial lymph space. Throughout the cord this infiltration is almost universally found especially in the ramifications of the anterior spinal vessels and the blood vessels of the pia. It is never as marked in the cerebrum and cerebellum and unless careful search is made may not be found. It is most marked in the pial vessels and in the larger vessels deep in the central white matter and in the peduncles. It is often absent in the vessels of the cortex, occasionally being noted in a cortical branch of a meningeal vessel or in some of the larger cortical vessels. In the vessels of the gray matter I have never observed more than a single layer of scattered cells in the Virchow-Robin space. This is a marked contrast to the vessels of the cord where many layers of densely packed cells is the usual finding.

II. Tissues of Ectodermal Origin.

In the substance of the spinal cord the gray matter is the part almost exclusively affected. In the cerebellum there are changes in both the gray and white matter,

but those in the latter are the most marked and probably give rise to the disturbances of cerebellar function noted in the clinical cases.

The changes in the gray matter consist of focal areas of cellular accumulation in the molecular layer. These focal accumulations do not differ from those seen in the gray matter of the anterior horns except that they are less dense and do not seem to bear any relation to the blood vessels or nerve cells.

The stellate and small cortical cells in the neighborhood do not show any particular alterations. The Purkinje cells in the infiltrated area may show degenerative changes but those in the immediate neighborhood are not particularly altered. The character of the infiltrating cells is the same as the character of those found in the cord; viz., lymphocytes, polyblasts, amœboid neuroglia cells, plasma cells and a few polymorphonuclear leucocytes.

There is considerable edema in the affected areas. The Purkinje cells throughout the cerebellum may show slight acute cloudy swelling, but are usually normal. This cloudy swelling is not more marked than would be expected in any acute infectious process.

There are no changes in the central limb of white matter of the folia or of its finer ramifications, but in the deeper white substance of the peduncles definite alterations are seen. These consist in areas infiltrated with cells like those described as occurring in the gray matter except that

the cells are frequently more densely collected together, and these areas are not as widely scattered as they are in the cortex. Hemorrhages are sometimes observed in the central white matter and may be present in the focal areas of cellular infiltration. I have not observed changes in the dentate nucleus.

From the pathological findings in the cerebellum it would appear that the virus of anterior poliomyelitis does not have the peculiar affinity for the nerve elements of the cerebellum which it shows for the motor cells in the cord, and that the symptoms referable to the cerebellum are produced by interference with the function of its connecting nerve tracts and not by destruction of its inherent nerve elements.

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THE CEREBRAL CORTEX IN ITS RELATION TO SENSATION AND MUSCLE TONE

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The patient, M. R. (24998), is a girl, eight years of age. Date of first examination, May 15, 1919, after which several examinations were made, the last being June 5, 1919.

HISTORY. Present Illness.—The principal complaints are the seizures described below, and a lack of use of the right hand and lameness of the right leg. These have existed practically since birth.

The patient was born Thursday, July 1st, and on the following Monday she had convulsions which lasted two days and two nights. During these convulsions the top of her head was sunk in, returning to its normal contour when they were over. The convulsions consisted of a turning of the head and eyes to the right and convulsive movements of the limbs, especially on the right. No facial movements nor tongue-biting were noticed.

The convulsions recurred once a month or so until she was about six years old. Since then they have become less frequent and milder in type, consisting of unconsciousness, a turning of the head and eyes to the right and a general stiffening of trunk and limbs. They continue about ten to twenty minutes and are followed by a severe pain in the head lasting a couple of hours. She sometimes vomits during them or "tries to." Her lips get very red before an attack.

When a baby, the right fist was always closed. This lasted until she was about four years old.

Previous History.—Birth: She was born at eight months and has a similar twin sister, the patient being first. Instruments were used, and while the mother did not notice any marks the doctor told her that he expected something would be wrong

with the patient, apparently meaning a brain injury. There was no cyanosis nor asphyxia, but apparently there was with the sister, who was blue. Her weight was 4 pounds, her sister's, $3\frac{1}{2}$ pounds.

Development: Both were breast fed. Her first teeth came at one year, her sister's at nine months. She began to walk at nearly three years, her sister at eleven months. She began to talk, "mama, papa," at about one and one-half years. Her sister "talked long before she did," and "is smarter." No impediment or difficulty of speech was noticed. Control of sphincters apparently was established in both about the same time. Both are subject, though seldom, to nocturnal enuresis.

Habits and General Physiological Status, negative.

Diseases, Injuries, etc: There is no complaint as to vision or hearing. Both sisters had scarlet fever at seven years of age and mumps with measles at six years.

Family History.—The twin sisters are the only children. No miscarriages nor stillbirths. Both parents are living and well.

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—The right hand is carried with the fingers nearly extended—slightly palmar flexed, the forearm is pronated and slightly flexed. There is a tendency when recumbent to dorsiflexion of the big toe with pes equinus and cavus on the right

Gait. There is a right limp and a slight right toe to heel steppage.

Coordination.—Equilibratory: This shows no disturbance.

Non-Equilibratory: This appears to be normal except as interfered with by weakness or contraction. The heel-to-toe test was done rather poorly on the right and

left, but was not ataxic. Touching the thumb with each finger of the same hand in succession is done poorly with the right hand with eyes open and closed. Closing and opening the right fist is done poorly with the eyes open and cannot be done with eyes closed.

Abnormal Involuntary Movements.—None were noted. The patient is restless and appears to tire mentally easily while being tested.

Reflexes.—Deep: These are not very

ited in extent. Flexion of the right fingers is weak and limited, extension is weak, abduction and adduction are weak and limited. The fingers when extended are hyperextended, producing the familiar "saddle-backed" appearance (Fig. 1).

There are slight and somewhat doubtful pareses of the various movements of the right thigh except rotation outward, weakness of which is more pronounced. Flexion of the right leg shows weakness, extension is normal. Plantar flexion of the right foot



FIG. 1.



FIG. 2.

well marked. The right patellar is perhaps slightly greater than the left. The uncrossed tibio-adductor is only present on the left. There is no crossed patello-adductor and no ankle clonus.

Superficial: The abdominals are active and equal. There are a right Babinski, Chaddock and doubtful Schaefer.

Muscle Strength.—The head movements appear to be of normal strength and equal in opposite directions. The right shoulder movements are limited and the arm can only be raised to a moderate extent. The right forearm cannot be supinated and its pronation is weaker than on the left. Volar flexion of the right hand is weaker, dorsiflexion cannot be performed, abduction is weak and adduction is lim-

is somewhat weak, dorsiflexion cannot be performed. Abduction and adduction of the right foot are slight. Plantar flexion of the right toes is either limited or absent and their dorsiflexion is limited.

Muscle Status. Volume.—The following measurements, made by Dr. Charles H. Jaeger, show diminution of bone as well as muscle. Arm: right biceps 7, left $7\frac{1}{2}$ inches; right elbow $6\frac{7}{8}$, left $7\frac{1}{8}$; two inches below elbow, right $6\frac{1}{2}$, left $7\frac{1}{4}$. Lower extremities: right thigh $13\frac{7}{8}$, left $14\frac{1}{4}$; right knee $10\frac{3}{4}$, left 11; right calf $9\frac{1}{8}$, left $10\frac{1}{4}$; distance from right anterior superior spine to right internal malleolus $25\frac{1}{2}$, on left 26. The right foot is $\frac{5}{8}$ inch shorter than the left, circumference about the middle $\frac{3}{8}$ inch less on right than left.

Semicircumference of trunk from middle of upper part of sternum around arm to spinous process of vertebra is 2 inches less on right than on left.

Consistency: The right hand and fingers are soft.

Tone: The right fingers are hyperextensible at all joints, least so at the metacarpophalangeal, the ring finger less than the others (Fig. 2). The left fingers are possibly somewhat hyperextensible. Volar extensibility of the right hand is greater than the left but dorsal extensibility is less, showing contraction. There is contraction of the pronators of the right forearm, of the plantar flexors and adductors of the right foot. There is a slight hypertonus seen in flexion and external rotation of the right leg.

Abnormal Associated Movements.—The right hand shows associated movements with the left hand, e. g., clenching the left fist unavoidably produces a clenching of the right. In the latter the thumb is always between the index and middle fingers. Opening the left fist is not associated with opening the right.

GENERAL SENSORY.—The cooperation was not good enough for protracted or exacting tests. The patient fatigued easily and became restless.

Touch.—Acuity: There is a marked diminution or even absence of light touch over the palmar tips of the right fingers, less diminution over the dorsal aspects and over the palm. Careful testing with very light touch or rather very light pressure with a blunt point made it probable that there is also a diminution on the right lower extremity and probably over the right trunk. No diminution on the right face could be established. Repeated light stimulations of the palmar surfaces of the right fingers and hand not sufficient in themselves to elicit a response finally cause an itching which the patient noticed though she had not apparently noticed that she was touched.

Localization: This is markedly dimin-

ished, i. e., inaccurate on the right fingers and hand, and is also poorer on the right leg than on the left.

Discrimination: This is also much diminished on the right fingers, in fact sensation was too much diminished to give good results. The toes, right and left, gave ambiguous results. The right forearm was simply guessed, the left gave consistent results at 2 to 3 cm.

Textures: Smooth paper and several grades of sandpaper were used and showed a diminution of appreciation on the right hand. There was evidence also, when care was used, that this is true of other parts, even, to a very slight degree, of the trunk.

Stimulation with faradic current apparently shows a slightly higher threshold on the right side excepting, perhaps, the anterior thigh and leg. Subsequent observations on the fingers and hand made this somewhat doubtful.

Pain.—This as far as tested shows no change.

Temperature.—Cool and warm were used. These show some diminution, i. e., wrong naming at times as compared with the left, on the right fingers and there was some evidence that this extended to other parts, even to the trunk.

Vibratory.—There is possibly some diminution on the right fingers. This could not be established elsewhere, though there were some indications on the right leg.

Pressure.—Light pressures appear to be affected similarly to touch.

Muscle-tendon.—This is diminished in the right fingers, probably in the right hand and also, to a less extent, in the right big toes and foot, possibly also in the right leg. The right fingers appeared to show paralysis (and apraxia?) rather than ataxia.

Stereognosis.—This appears to be practically absent in the right hand. Such obvious objects as a rubber ball and knife were not recognized. A flat hexagonal piece of iron (a weight) was not recognized and was said to be soft, all that was felt was that it was cold. When placed in

the hand on a subsequent occasion it was correctly named, evidently on account of its coldness.

Barognosis.—This could not be tested well on account of the pronated position of the hand. Various changes in weights placed on the backs of the fingers, which rested on a cushion, were obviously not identified well, or at all, on the right.

In general, the results obtained on the right, at least on the right fingers, hand and forearm, show greater variability than on the left. The easy fatigableness of the patient has already been noted. Whether this fatigue and lack of concentration was more marked while testing the right side is not certain though one rather got the impression that this was the case and it was stated to be the case by the patient. The efforts of the patient to supplement her deficient sensibility by peeping, etc., were quite persistent.

CRANIAL NERVES. *Optic, etc.*—Fields of vision, fundi, reaction of pupils to light and to convergence are all normal. The eye movements are normal except there is an insufficiency of convergence and there is some diplopia. There were some slight nystagmoid movements with eyes to right and left.

The Facial.—Bitter was tasted well on right and left. Sweet gave ambiguous results on both sides. There is a slight lower right facial paresis and there is drooling on the right.

The Vagus, etc.—The uvula is normal in position and moves normally during phonation.

The Hypoglossus.—The tongue can be moved better to the left than to the right. This movement is evidently performed by the intrinsic muscle of the tongue.

MENTAL STATUS.—This appears to be normal, excepting the fatigableness and restlessness already noted.

CRANIAL MORPHOLOGY.—Nothing abnormal was noted.

SYSTEMIC. *Tegumentary*.—There is a

slight vasomotor weakness of the right arm and hand, which tend to be cooler than the left. The lips are usually very red. This is true also of her sister.

DISCUSSION

That we are dealing here with a hemiplegia, probably due to a birth injury is evident. With the exception of the Babinski the reflex changes are very slight, but this deviation from the classical picture is not perhaps so infrequent as one would expect from textbook descriptions. The two peculiarities which are more infrequent and make the case of interest, are the hypotonia of the right hand and the sensory disturbances.

Head, in his article on "Sensation and the Cerebral Cortex" (*Brain*, Vol. XLI, part 2), has paid considerable attention to this hypotonia. According to him we may have:

(1) A hypotonia associated with want of special appreciation and without a motor defect. Such cases will naturally show an ataxia but finger movement may be well performed with the eyes open.

(2) Cases where the movements are clumsy with the eyes open and where reflex changes point to an interference with the pyramidal system. Here there may be no apparent hypotonia.

(3) The usual spastic condition where the pyramid is severely affected.

"(4) Where severe lesions of the cortex and subcortical tissues may cause a complete flaccid paralysis, especially in the early stages. Here the fingers are atonic because both voluntary movement and static tone are destroyed by the injury."

He also points out that the paralytic loss of tone may in certain cases exist under cover of general spasticity of the limbs, as is shown by hyperextensibility when the spasticity is overcome by gentle and steady pressure. We may add that the peculiar hyperextensions assumed in athetosis are also obviously related to this phenomenon. According to the above, briefly,

injury to the cortical mechanism for the appreciation of position and movement produces hypotonia, pyramidal injury produces spasticity, injury to both in varying degrees may produce varying tonic changes and severe injury to both may produce atony, especially in the early stages.

According to this, Head points out, "recovery of motor power without coincident restoration of postural sensibility should lead to the gradual appearance of hypotonia in the affected fingers." Head studied a case of this kind in which, eleven months after the injury, with improvement of movement, hypotonia appeared. The case we have before us obviously comes under this category. According to the history the child's right fist was clenched for several years, but since then there has been a considerable return of movement and appearance of hypotonia, appreciation of position and movement remaining seriously affected. In the present case we have a beautiful topographical transition from spasticity to hypotonia, the former existing in a high degree in pronation of the forearm, to a less degree in dorsal extension of the hand, changing to a hypotonia in volar flexion of the wrist and extension of the fingers. The associated fist-clenching appears to be a remnant of the old spastic fist-clenching in spite of the supervening hypotonia.

Head enters into explanations of the cause of this hypotonia but does not discuss it from an anatomical point of view. Whatever the value of his remarks in illuminating this condition the fact remains that the hypotonia is a condition of muscles due to cerebral conditions and there must be an anatomical nexus between the two. If the hypotonia is produced by cortical lesions of certain parts outside the motor area, perhaps, but whose only pathway to the muscles is the pyramidal system, why should the pyramidal lesion itself produce a contrary effect? It is true that lesions of afferent paths to a given center produce different effects from those due to injuries of efferent paths; yet it is

difficult to see why a hypotonus should be transformed into a hypertonus on passing over from afferent to efferent. The most natural assumption is the postulation of another efferent path beside the pyramidal one, and the first path to suggest itself is the pallio-ponto-cerebellar one and possibly others to various basal nuclei. In this connection there may be recalled the atony described by Foerster and probably due to frontal lobe lesions. In cases like the one before us the changes are seen in distal parts, parts which are preëminently the parts performing active, complicated skilled movements. The proximal and trunk musculatures, more purely involved in maintaining posture of the body, and in performing more elementary acts such as walking, are here comparatively little involved. Perhaps in the latter, mechanisms are concerned which are more frontally situated and which use to a great extent such an efferent path as the pallio-ponto-cerebellar one, the pyramidal being less important.

It is interesting to note here, in the cortex, a hypotonia associated with injury to a proprioceptive mechanism; and when we consider conditions seen in lesions of posterior roots, posterior columns, afferent cerebellar paths, the cerebellum itself, etc., it would seem that we might generalize and say that lesions of the proprioceptive, or at least the afferent proprioceptive mechanisms always produce hypotonia. Head, however, appears to consider the particular hypotonia discussed above as due to cortical lesions and we have seen a case of loss of sense of position and movement of fingers and hand without any considerable paralysis and yet without marked hypotonia.

Head, in the article above referred to, has also added to his previous very valuable contributions to the sensory mechanisms. He is not in these studies dealing primarily with the anatomical side but has used his material to study the various dissociations and rearrangements of various categories of sensation, at various levels. In his familiar peripheral nerve work the dis-

sociation between deep and cutaneous sensation, the former including impulses used in localization and the latter those in discrimination, is brought out, and likewise the much discussed dissociation, seen in nerve regeneration, between protopathic and epicritic sensation. In the cord we have the familiar rearrangement into the paths of pain, temperature, touch, those touch impulses necessary to touch discrimination, and finally those impulses necessary to the appreciation of position and passive movement. In the brain stem the two latter become dissociated and as the thalamus is approached other rearrangements may begin leading to the termination of touch, pain, heat and cold in separate thalamic end-stations. In the "essential organ" of the thalamus we have in a sense the termination of those portions of the sensory paths carrying impulses giving rise to affective states, while those destined to undergo the associative activity of the cortex are simply relayed and pass onward to the cortex in the thalamo-cortical radiations. The former comprise nearly all of pain, the larger or a considerable part of heat and cold and a smaller portion of touch and vibration, while the latter comprise a smaller part of heat and cold, a larger part of touch and all of position and movement. The cortex itself is, of course, the associative memory mechanism for the stimuli which reach it. Its activity, in relation to sensation, is manifested, according to Head, in "(a) recognition of special relations; (b) a graduated response to stimuli of different intensity, and (c) appreciation of similarity and difference in external objects, brought into contact with the surface of the body." Lesions of the sensory cortex would manifest themselves then by disturbances of appreciation of position and movement, involving, as this does, an unavoidable comparison with previous position; by an inability accurately to discriminate intensities, such as different degrees of warm and cool; inability to obtain touch thresholds within reasonable limits and inability to

distinguish different sizes, forms, weights and textures. The loss is the more severe the more complicated the association, i. e., the more factors compared. The cortex is the mechanism of attention and in lesions of the kind under consideration a diminution of attention in the sphere involved and greater attendant fatigableness are other aspects of the same loss or diminution of function. Various introspective changes may also be noted.

It is not the purpose here to give a complete summary of Head's results, but he points out that some tests depend upon more than one of the above fundamental qualities, e. g., the compass test may depend upon both tactile intensity and two-dimensional spacial relations. Furthermore, tests obviously also differ in respect to their relative appeal to thalamus and cortex.

If we glance at what are commonly admitted to be the leading features of thalamo-pallial arrangements an anatomical basis is suggested for the above, but certain difficulties also become apparent.

The thalamus, as is well known, consists of two parts, an outer or lateral part connected with the cortex by the thalamic radiations, the latter, according to prevailing views, consisting of both thalamo-cortical and cortico-thalamic fibers. The lateral thalamus also receives the afferent paths of general bodily sensibility, and the further continuation of this path to the cortex is by a portion, but only a portion, probably, of the thalamo-cortical fibers. These latter fibers proceed, probably, to the sensory cortical areas.

The other, internal or medial, portion of the thalamus is not connected directly with the cortex and is supposed to consist of neurons with shorter axons which associate various parts of the medial thalamus with each other or associate the medial thalamus with the lateral thalamus. The medial thalamus probably also receives fibers from the lateral thalamus.

The thalamus is also connected with the corpora striata.

In view of the above, it is natural to assume, as Head and others have done, that the sensory paths after their termination in the lateral thalamus have two continuations, one by thalamo-cortical fibers to the sensory cortex, there to undergo the associative, discriminative rearrangements performed by the cortex, and the other, probably by short neurons, to the medial thalamus which is the center for affective changes. The remaining thalamo-cortical fibers may be conceived as conveying stimuli which when entering the cortex give the general affective tone which accompanies, or is a part of, all our conscious activities and is a synthesis of a great variety of organic as well as peripheral stimuli not well localized nor discriminated. These affective states, viewed objectively, appear to have much the same general function as the more concrete ones, such as pain from a pin prick; i. e., they probably lower the cortical threshold, activate the cortex and reënforce the vigor of the response. The latter may be misdirected, too generalized, etc., when the discriminative activity of the cortex is not sufficiently in play; but the result of a lack of thalamic influence is equally evident in the lack of adequate response seen in cases of depression and apathy where interest is lost and where, without any lack of discriminative (mental) capacity, reactions are not completed. The thalamo-cortical fibers under consideration would obviously reënforce those by the direct route and give a more intense cortical response.

It is evident from the above, that while it may be true that the cruder affect reactions may not get beyond the thalamus and that organ may alone be necessary for their "awareness," the more widely diffused though often less intense affect tones of all our activities must be regarded as participated in by the cortex. They are too many and varied and too closely connected with the discriminative activities themselves to be regarded as purely thalamic affairs. It must be remembered, too, that when the thalamic patient *speaks* of

his abnormal sensations he is using his cortex and there must be a thalamo-cortical nexus to perform this reaction.

The distinction between discriminative and purely affective reactions is of course one of objective observation; the latter are more diffuse, more generalized and involve usually a greater expenditure of effort. They are not so purposeful and well directed. The former probably reach the lower motor nuclei principally by the pyramidal system though their proper performance depends also on accessory systems which ensure a proper postural framework on which pyramidal reactions take effect. Do the affective reactions require another path? It is obvious that if some of them do not pass beyond the thalamus to the cortex, such do require a non-pyramidal outlet; and certain phenomena exhibited by pseudo-bulbar palsy, certain tremors, athetosis and chorea would seem to demand this, as well as the overreactions shown in thalamic lesions. These are here lumped together because in a large way, as pointed out in a previous paper and as also pointed out by a number of other writers, they show many similarities in form and in their relation to affective states.

We may, regarding the overreaction in a thalamic lesion, take the view of Dr. Joseph Byrne. According to this view, if I understand it correctly, there is no necessity for such a path from the forebrain centers. If stimuli do not reach the discriminative cortical mechanism the inhibitory effect of the latter upon lower centers is not exercised and we have a diffuse overreaction such as may be seen even in cord transverse lesions. This view, however, hardly removes the necessity for such a path in order to explain the marked effect of such cortically initiated impulses as voluntary movement, of effort without voluntary movement, as well as of emotional states, upon various tremors, chorea and athetosis. Another objection is that it does not explain well the function of the cortico-thalamic fibers.

Head and others regard the cortico-thalamic fibers as acting in an inhibitory way upon the purely thalamic mechanisms. If there be no other thalamic outflow than to the cortex, the cortico-thalamic fibers would then simply act as a dampener so to speak upon the latter in some way—a rather peculiar circular or reciprocal mechanism. If there be another thalamic outlet their action becomes more intelligible.

It is probably premature to go far in a discussion of this question until we know more than we do as to the non-medullated fiber paths existing in the central nervous system, not to mention the further, unknown, connections of such gray masses as the corpus subthalamicum of Luys and the substantia nigra. It may be well to point out, however, that we apparently do have a connection by way of the corpus striatum which, as already mentioned, probably receives fibers from the thalamus and in turn gives rise to descending systems via the red nucleus, probably also the corpus subthalamicum and possibly by other routes. Such a thalamo-striate path might, when uninhibited by discriminative cortical activities, play a part at least in the abnormal movements above referred to. This "inhibition"—or, better, integration of two influences—may, it must be remembered, take place not necessarily at the thalamic level but perhaps at the cortical level or at the lower motor neuron level. The above path is apparently in agreement with the views expressed by J. Ramsay Hunt, although the production of such antagonistic phenomena as those shown by paralysis agitans (loss of associated movements) on the one hand and by athetosis (excessive associated movements) on the other by lesions of different parts simply of the same mechanism, certainly presents difficulties.

As a result of the above, if we view the cortex on the efferent side, there should emerge from it three streams of impulses, one which actively contracts groups of muscles in such a way as to produce definitely limited, purposeful, skilled acts; an-

other which acts upon the general postural, tonic muscular mechanism, and a third which regulates the affective, including splanchnic, as distinguished from discriminative reactions. The first would be largely the pyramidal system, the second largely the pallio-ponto-cerebellar and the third the cortico-thalamic. Perhaps when these mechanisms are better distinguished and understood such phenomena as katatonias, stereotypy, schizophrenia, certain manic-depressive states, etc., not to mention hysteria, will receive some neurological illumination even though they be not all so-called organic affections.

While in theory the distinctions made by Head between the effects of interruption of the thalamo-cortical system and the effects of injury to the associative cortical mechanism are clear and seem obviously based upon what must be the nature of the cortical apparatus, it must occur to everyone that in practice the separation will not be so easy, at least as far as qualitative distinctions are concerned. This is owing to the fact that the thalamo-cortical fibers must obviously enter the cortex to encounter the cortical associative neurons; and it is difficult to see how there can be a dissociation between the two unless of course some systemic disease intervenes in our behalf. As a matter of fact, though, Head has shown cases where there was at least a sufficient dissociation to demonstrate his point. We have, of course, to distinguish between lateral thalamic lesions, capsular lesions, subcortical lesions and cortical lesions; and it must be admitted, it seems to the writer, that leaving out of account the greater liability of lesions in certain locations to implicate certain other structures, such as the pyramid, the distinction is difficult. There does not appear to be any *a priori* reason for any qualitative difference between a lateral thalamic, a capsular and a subcortical lesion or even, perhaps, a cortical lesion, as shown above. In the lateral thalamic lesion we have usually the increased affect. This may be due to the fact that a lesion

here will usually catch a larger proportion of the thalamic radiations to and from the cortex. I see no other reason for the presence of this phenomenon in thalamic lesions only, inasmuch as in capsular, subcortical and cortical lesions the cortical inhibition should be equally lost due to the cortex being cut off. As a matter of fact in Head's papers the distinctions between these levels are not entirely clear. In all cases we are confronted with the distal distribution and the greater loss of position and movement, next, loss of tactile responses and lastly no loss of pain. There may be topographical distinctions, e. g., the deeper the lesion the more profound the sensory loss and the greater tendency to a hemiplegic distribution. The anatomical reason for this latter is obvious and is mentioned above.

The qualitative distinction between cortical and subcortical lesions should, as already stated, be the presence in the former of a loss of association, i. e., of ability to compare those sensory stimuli which have passed the cortical threshold both in time (different intensities, change of position, etc.) and space, this loss being superadded to any mere raising of the threshold. Head's cases appear to show this in some respects, but they also appear to illustrate the difficulty of distinguishing in practice between the mere diminution of sensibility and the diminution of comparison. He speaks, for example, of the profound affection, in cortical lesions, of the sense of position and passive movement and speaks of this as being due to the fact that there is inevitably involved in this "sense" a comparison with previous positions, the cortex thus creating a continuous series of plastic poses ("schemata"). Is, however, the sensory loss of this kind when due to a cortical lesion different from that produced by a lesion of the posterior columns of the cord where of course the comparing cortical mechanism is not affected? The same considerations apply to tactile discrimination of points. All the varied cortical tests should be applied in cases of pos-

terior column lesions also. Apropos of cord lesions, may not the peculiar homolateral hyperesthesia sometimes seen in lesions of one-half of the cord be due also to removal of cortical discriminative activity and thus similar to that seen in the thalamic syndrome?

While the present case in some respects, e. g., the tendency to a hemiplegic distribution, would seem to present evidence of being subcortical, I believe it is probably, at least in part, cortical. A series of careful quantitative tests could not be carried out such as would be necessary to decide solely on the basis of the sensory changes; but the markedly apical distribution, the fact that tactile localization, tactile discrimination and stereognosis were all apparently affected out of proportion to the mere sensory diminution, the variable results as to mere perception of simple stimuli (change of threshold), the fatigableness of attention, all point to a cortical lesion, although with some reserve on account of the preceding criticisms. Quite interesting was the phenomenon, noted several times, of a response to very light touch being obtained only when, after repetition, the part stimulated began to itch. Here a summation of stimuli appeals first to the thalamus apparently and then the cortex is aroused. The hypotonia, the character of the injury and the seizures also indicate a cortical lesion. That the case is due to a birth injury appears to be fairly clear. We have here, it is interesting to note, an identical twin without defect, a fact which excludes nearly all other etiological factors, whether hereditary or an acquired disease.

Regarding localization, Head places the lesions producing loss of sensation of the cortical type in the "pre- and post-central convolutions, the anterior part of the superior parietal lobule, and the angular gyri." He says:

"(a) Injury anywhere within the sensory area on the hemisphere is liable to cause some loss of appreciation of spacial relationships in the hand or in the foot. But the farther forward it lies within this

region, the grosser and more extensive is the disturbance shown by the spacial tests.

“(b) The power of recognizing differences in weight and shape is most severely and most extensively affected by a lesion which falls over the post-central convolution.

“(c) Those tests which depend mainly on due recognition of stimuli of graduated intensity are more likely to be disturbed when the injury falls upon the posterior and inferior portions of the sensory area. Any lesion lying mainly within a zone which occupies the foot of the pre- and

post-central convolutions, the angular and supramarginal gyri and the superior parietal lobule, is liable to be associated with disturbances of tactile sensibility. If this is severe and extensive, the patient may be unable to distinguish various degrees of warmth about which he has no doubt on the normal hand.”

The disturbances in the present case fall more under (a) and (b) than (c) though the latter are also to some extent affected. The association with motor disturbances also, of course, show that the lesion extends anterior to the fissure of Rolando.

A CASE OF SACRAL CORD INJURY AND A SUBSEQUENT UNILATERAL SYRINGOMYELIA

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The patient, P. C., No. 25526 (E19247) is a man sixty years of age, single. Date of admission to the Vanderbilt Clinic, July 7, 1919. Occupation, formerly a derrick man, now a porter.

HISTORY. Present Illness.—The complaints are paralysis of the bladder and paresis of the rectum, loss of sexual function, numbness of the whole left side except the face, poor use of the left hand and a sore on the sole of the right foot.

January 30, 1890 or 1891, he fell from a building and was struck in the small of the back. He was unconscious five or six hours; was taken to a hospital and remained there until June 10th. He did not work for ten months, chiefly, apparently, on account of pain in the back. He did not notice any lameness or numbness after the accident, but had loss of sexual power and loss of control of bladder and rectum. He has had this trouble ever since, especially affecting the bladder, but six years ago the latter became much worse and since then he has worn a rubber bag.

In 1897 he suddenly felt a numbness in the left arm. About ten days after this his left hand became affected so that he could not shave himself, he could not—and cannot now—hold small things well in this hand. About a couple of months later the numbness of the left side was noticed. Apparently there has been no marked change in the numbness since 1897. The arm sometimes feels hot and sometimes cold, as well as numb. There is also some complaint of pain in the neck. There is no history of accident between 1891 and 1897.

Four years ago a sore appeared on the sole of the right foot. It contained a watery substance but no pus. It would heal and then break out again. Last December gangrene of the right second toe set in and

the toe was amputated in St. Francis' Hospital. Three weeks after leaving the hospital, the last of February, the old sore broke out and after unsuccessful treatment by a doctor he went to the Presbyterian Hospital about the middle of May. There he was massaged and baked and much improved. On account of the anesthesia he was sent to the Vanderbilt Clinic for examination by Dr. Frank Meleny, who thought he might have syringomyelia. The Monday before coming to the clinic a blister came on his heel when being baked, though those before and after him did not blister. He did not feel it.

He has also been troubled, but apparently not very severely, with pains in the right knee, the left lower back and the left back of the neck, some of them for the last ten years or so.

Previous History.—Habits and General Physiological Status: These are negative except as already mentioned.

Diseases, Injuries, etc.—These are also negative except as already mentioned, and except that he was severely burned on the left arm and face when a few years old. There are no disturbances of vision or hearing, no headaches, no vomiting and no history of diseases.

Family History.—His father died at ninety-five, his mother at eighty-four.

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—None were noted except a tendency to incline the head to the right.

Gait.—This is normal except a limp due to stepping on the right sore foot. He can walk well on toes and heels.

Coordination.—The patient is right-handed.

Equilibratory: Standing with feet together and with eyes open and closed is

practically normal; there is a slight swaying with the eyes closed

Non-equilibratory: The finger-to-nose test is performed well with the right hand, with the left there is a slight tremor and slight occasional ataxia. The finger-to-finger test is the same. Touching the thumb with each finger in succession is done better with the right than with the left. Pointing and pass-pointing is done accurately excepting a tremor on the left. The heel-to-knee test is done well. The patient missed the first two or three times with the left and then performed accurately.

Skilled Acts.—The handwriting shows no abnormality other than the irregularities to be expected in a person not accustomed to doing much writing.

Abnormal Involuntary Movements.—There is a fine rapid tremor of the left hand. Occasional fibrillary twitchings of the interossei of the left hand were noted.

Reflexes.—Deep. The right biceps, and triceps are active, the radial slight, the left are less active. This may be due to a cicatrix, the result of the old burn mentioned above which has produced a tendon-like structure extending along the left forearm to the thumb. The left patellar is active but the right is only slight on reënfocement. Both Achilles are absent.

Superficial.—The upper right abdominal is active, the left is absent. There is no Babinski on the left, the right was not tested on account of the condition of the foot. The anal and cremasteric reflexes are absent.

Muscle Strength.—This is normal in all parts except the right toes, the small ones showing loss of movement and the right big toe being weaker than the left. Dorsal flexion of the right, foot and inward rotation of the right thigh seemed to show some slight diminution compared with the left. The left hand and fingers are somewhat weaker than the right, but it is doubtful whether this is greater than what would be due to its being a left hand and one not used much and somewhat crippled.

Muscle Status.—There is apparently

some atrophy of the left first interosseus, but this appearance may be due to the action of the cicatrix mentioned above.

GENERAL SENSORY. There is a great diminution or loss of pain and temperature and a diminution of touch acuity over the whole left half excepting the face. Towards the head, however, in the upper cervical segments, there is less diminution and at the boundary of the trigeminus the above sensibilities apparently become normal; but, when compared with the right face, touch and pain at least are, according to the patient, less plain or sharp than on the right. There also appears to be a somewhat lessened diminution, or rather not a complete loss, of pain and temperature on the lower thoracic and first lumbar segments. Touch does not appear to show this change, in fact is, if anything, somewhat more diminished in the lower thoracic and first lumbar segments. Pain and temperature are lost on the upper lumbar segments, reappear to a slight extent on the lower lumbar and first sacral and again disappear on the lower sacral. Touch is only lost on the low sacral. There may be more irregular variations elsewhere which have escaped notice. For example, there appears to be less diminution of pain on the ulnar portion of the hand.

On the right side, pain, temperature and touch appear to be normal, or nearly so except on the 2d to 5th sacral segments where they are lost. This loss may extend partly over the first and is quite sharply marked off.

Touch discrimination is normal on the right finger tips but is lost on the left.

Touch localization does not appear to be much affected. Pressure is diminished on the left side. At times synchronous bilateral pressures on the trunk were stated to be only on the right side.

Vibratory is normal on the right side except the shin, ankles and toes, where it is absent, and the pelvis, where it is possibly diminished. On the whole left side there is a slight diminution except on the shin,

ankles and toes, where there is a greater diminution or complete loss.

Muscle-tendon.—The sense of passive movement is normal on the right except the toes and foot, where it is absent. The left side shows apparently, when carefully tested, a diminution in the fingers, a probable slight (introspective) diminution in the forearm and arm. No diminution could be established in the left lower extremity.

Stereognosis.—This is normal in the right hand and greatly diminished—nearly absent—in the left.

All forms of sensibility as far as tested showed diminution over the edematous area on the right foot and lower leg mentioned below.

CRANIAL NERVES.—*Optic.* The fundi were examined by Dr. Schoenberg and show some sclerosis and consequent slight paleness and indefiniteness of the discs. The pupils react well to light and convergence. The eye movements are normal except a slight convergence insufficiency. There is no nystagmus.

Trigeminus.—The corneal reflexes are present on both sides.

Facial.—The facial movements are normal. There is a slight facial tremor.

Hypoglossus.—The tongue movements are normal.

MENTAL STATUS.—This appears to be normal.

SYSTEMIC. Tegumentary.—Besides the ulcer on the sole of the right foot there are scars of trophic sores or ulcers on the glutei and the scars (from a burn in early youth) already mentioned on the left hand, forearm, arm and face.

At some of the examinations the patient showed a rash present on the shoulders and trunk to some extent on both sides, but on the abdomen confined to the left side and stopping abruptly at the median line. There are swelling and trophic changes of the right foot and lower leg.

Cardio-vascular.—A report from the Presbyterian Hospital stated that an x-

ray showed calcification of the blood vessels of the foot.

Skeletal.—The same report stated that the x-ray showed signs of atrophy of most of the bones of the foot and periostitis of the shaft of the tibia. An x-ray report by Dr. A. P. Evans states that "there is an old fracture of the first lumbar vertebra. This vertebra has been partially crushed and displaced to the right. Beginning ankylosis of the second, third, fourth and fifth lumbar vertebrae."

LABORATORY TESTS. Reports from the Presbyterian Hospital state that the Wassermann was negative to both antigens. The urine analysis showed specific gravity 1.020, reaction alkaline, albumin a very faint trace, glucose absent; microscopic, triple phosphate crystals and a small amount of pus.

DISCUSSION

It is obvious from the anamnesis, as well as on other grounds, that there are present two distinct and possibly disconnected phenomena, those presented by the lower extremities and the left anesthesia. The former are in part, at least, due to the trauma of 1891. An injury of the sacral cord would account for the paralysis of bladder and rectum and, if confined to the lower sacral segments of the cord, would not produce any paralysis of the legs. The apparent absence of anesthesia in the anamnesis can perhaps best be discussed later, but it may be pointed out here that the more profound anesthesia found on the right legs on the 2d to the 5th sacral segments and not extending as high on the left leg should be due also to this injury. Judging from the condition of the deep reflexes, the extent of the anesthesia and possibly from the trophic conditions shown by the right lower extremity this injury was more severe on the right than on the left.

Taking up the left anesthesia we note that it was noticed first in the arm, then

in the hand and later in the body. It is difficult to tell how much importance should be attached to its apparently later occurrence in the body because it might not be noticed there so promptly. In a general way, however, the clinical findings bear this out; for the sensory disturbances, some of them, at least, appear to be more marked (leaving the lower extremities out of account) on the arm and upper thoracic segments.

Regarding the qualitative character of the sensory changes, pain and temperature are affected the most and show the above distribution; i.e., the lower cervical and upper thoracic segments are most affected, the upper cervicals much less and the trigeminal region very slightly. The lower thoracics and first lumbar are somewhat less affected and the remaining upper lumbar are again more affected with a partial return in the lower lumbar and upper sacral and a final loss in the low sacral. On the right, pain and temperature are normal, or nearly so, except over the 2d to 5th sacral segments and possibly somewhat above this. There may be changes elsewhere on the right which would be shown by further examination, but there is certainly a marked difference between right and left.

Touch acuity is much less affected than pain and temperature on the left, and shows perhaps less variation in different segments. Touch discrimination shows greater change than touch acuity but localization is not much affected. On the right touch is lost, with pain and temperature, over the sacral segments mentioned above.

Vibratory again shows a slight left diminution except on the lower extremity where it shows a great diminution or loss. It is also lost or diminished on the right lower extremity.

Muscle-tendon sensibility shows a slight left diminution, especially in the fingers, perhaps doubtful elsewhere. In the right lower extremity it is lost.

Leaving out of account, for the present,

the lower segmental changes, how do we account for the left anesthesia? A lesion of the right lateral white column of the cervical cord would of course account for the left loss of pain and temperature and diminution of touch acuity, but it would not account for their shading off in the upper cervical and probably in the lower thoracic segments, nor would it account for the changes in touch discrimination, vibratory and muscle-tendon sensibility which are obviously due to a slight *left posterior* column lesion. If we put the lesion in the left cord, especially in the gray matter, how do we escape a bilateral loss of pain and temperature? This latter objection can be avoided, obviously, by placing the lesion in the left posterior gray of the cord. In this position the terminations of the pain and temperature root fibers in the gray, their synapses with other neurons or the secondary neuron bodies themselves may one or all be affected, the pathway from right to left remaining unaffected. As soon as we place the lesion in the gray, however, we must extend it longitudinally over as many segments as those over which the sensory loss in question extends, in this case involving all the segments of the cord. Such a lesion can practically only be due to a syringomyelic process or possibly a hematomyelia or hydromyelia. The slight hypesthesia of the left face would be due to the descending root fibers of the trigeminus in the cervical cord being affected.

Regarding the posterior column changes, we have two alternatives: there may be a slight lesion confined to the portions adjacent to the dorsal gray column. This might produce deep sensory changes varying in different segments. On the other hand there might be a lesion affecting the whole column which obviously would produce changes in all segments below the lesion. There is obviously not a *total* transverse lesion of the posterior column and the deep sensory changes are not marked enough to decide the question, especially in view of the lower complication. The

simpler explanation would apparently be the first. The apparent dissociation between vibratory and muscle-tendon sensibility on the left lower extremity is peculiar.

The tactile changes possibly present some difficulties. One would expect, perhaps, a still more profound change due to the fact that both dorsal gray and column are affected—in fact the rather surprising marked astereognosis may be due to this. It must be remembered, though, that the posterior column changes are rather slight probably.

With the above dorsal column changes a diminution of the left arm jerks would a priori be expected and this inclines one to the opinion that their apparent lessened activity in the left arm may be a neurological and not a purely traumatic phenomenon. In fact one would expect their abolition with a gray lesion as above postulated. The slight ataxia shown by the left arm would also naturally be due to the posterior column changes. We are prohibited from having recourse to a lesion of the spinocerebellar tract on account of the fact that there are no signs of pyramidal involvement and it is difficult to see how the spinocerebellar tracts could be reached without the lesion traversing the pyramid.

The occasional fibrillary tremor I am inclined to believe is significant of an extension of the lesion to the cervical ventral gray.

When we examine the lower lesion, some difficulties are encountered. We have, on the right, a loss of the Achilles (L_5-S_2), a diminution of the patellar (L_2-L_4), a loss of muscle-tendon sensibility in the foot and leg but no loss of strength in the same parts (excepting the toes and other doubtful parestheses) and a loss of vibratory sensibility in the feet and legs, involving probably L_4 or L_5-S_2 . Regarding muscle-tendon sensibility, passive movements are presumably perceived by the movements of both protagonists and antagonists and the movements of the leg and thigh may be

therefore perceived normally on account of the presence of the higher segment muscles of the anterior thigh. The articular surfaces also are probably innervated by the higher lumbar segments as well as lower. It is still difficult to see, however, why the patellar reflex is affected. We might explain this by assuming a gray lesion extending somewhat further upwards than the rest of the lesion, a not improbable assumption. If this is the case why are not the left arm jerks obliterated by the gray lesions there, a point already raised above? Even with the above explanation, the deep sensory and reflex changes appear to extend higher than is indicated by superficial sensation and muscle strength, which latter should apparently be affected more. This can be explained by assuming the lesion extends further upwards on the dorsal aspect of the cord.

It is rather surprising that the standing tests reveal such slight or even doubtful changes in equilibratory coordination.

We thus have a lesion involving the lower sacral segments of the cord and probably extending above this on the right dorsal half, furthest upward in the dorsal gray. Partly superimposed upon this is a long lesion of the left dorsal gray extending through all the segments of the cord but most marked in the lower cervicals, upper thoracics and upper lumbar.

An interesting question is the possible causal connection between the trauma and the syringomyelia. Some points may be raised against there being any connection, such as the fact that the sacral lesion is more on the right, the syringomyelia largely or entirely on the left. Another point is the fact that apparently the latter began in the cervical and upper thoracic segments. The fact that some six or seven years elapsed before the syringomyelic symptoms were noticed and the great length of the lesion are decisive against the syringomyelic lesion being simply a hematomyelic extension of the trauma. The question still remains, however,

whether the latter might not have initiated the former pathologic process. The traumatic origin of syringomyelia has many adherents and it is easy to imagine, at least, how a cord trauma such as a crush would act upon the ependyma along an open central canal, possibly loosening the cells and initiating an abnormal proliferation. The points raised above are not decisive against such a process in the present case, nor indeed is it proven by any means that the trauma and syringomyelia did not overlap at the very beginning.

Another point is the occurrence of the trophic sores on the right foot twenty-four years after the injury, and eighteen years after the syringomyelia was first noticed. Their presence on the same side as the greater injury would indicate a connection

with that injury, but it is difficult to see why it should flare up so long after. Might it not be that the syringomyelic process *has* traveled downward and encountering the old injury has started these more recent trophic disturbances? It is interesting to note that they occur on segments representing the upper limit of the old injury. The increase in the bladder symptoms the past six years is also interesting in this connection.

Finally, the rather sudden onset and rapid spread of the syringomyelia, according to the anamnesis, as well as its lack of subsequent progress, are interesting and might be regarded as indicating some other process than a syringomyelia if we were not practically forced to accept the latter on other grounds.

AN ATYPICAL CASE OF MULTIPLE SCLEROSIS OF UNUSUAL ONSET

BY OLIVER S. STRONG, PH.D.

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NEW YORK CITY

The patient, A. Z., No. 25545 (E19413), is a woman thirty-six years of age, married. Date of admission to Vanderbilt Clinic, July 9, 1919.

HISTORY. Present Illness.—Her complaint is that she cannot use properly the left leg or the left hand which is subject to shaking, and that she has had some trouble with the right eye.

Last May when she got up in the morning she fell to the floor on account of the giving way of the left leg. There was no unconsciousness nor convulsive movements. It must be kept in mind, though, that the attack probably occurred during sleep. The leg was "like ice." There has been improvement in the leg since. This attack was preceded by pains in the head lasting two days.

About four weeks later the trouble with the left arm began, suddenly. She could not raise her arm and dropped a baby's bottle from her hand. At the same time there was a violent rotatory shaking of her left hand and forearm which lasted an hour or so. This was obviously in part at least a rest movement for she even tried to hold the hand still by pressing it between her knees but without success. The shaking lasted an hour or so. There have been subsequent attacks since, but less severe. The shaking has been both while the hand was in use and at rest. The weakness has improved. The left arm has also felt cold and she sometimes has chilly feelings but there is no history of fever. There has been some pain in the left shoulder and arm, none in the legs. Before this second attack she vomited twice.

A few days after the arm attack she began to have some trouble with the right eye. It was "like a veil over it" and would come and go. This has disappeared,

July 7th she had another attack of quite violent shaking of the left hand and forearm which lasted about half an hour. At the same time, about 8 p.m., a pain began, suddenly, above and below the right eye, which lasted about twelve hours. There has been no recurrence of the latter.

July 12th she complained of a pain for the first time on the left side of the forehead, over the eye and in the temple, and also of a "jumping" and pulling of the left arm. This was not like the shaking of July 7th.

Previously, in April, she had had some trouble with her hearing for a couple of weeks, apparently due to a cold.

Her husband says she was more noisy and nervous after the first attack than formerly. This has improved.

There is some complaint of "stomach-aches" since July 23d or so. She thought her periods were to be resumed, but this was not the case.

Previous History.—Habits and General Physiological Status: The patient sleeps well, her appetite is good, bowels irregular. There has been some loss of weight but apparently not significant. Since her trouble began she had had frequent and urgent urination with nocturia but no incontinence. Her first menstruation was at twenty years of age and she has had none since her pregnancy with her last child, born July 19, 1918.

Diseases, Injuries, etc.—There is no history of injuries. No operations. No history of convulsions, nor vomiting, except as mentioned. She has had headaches at her monthly periods for years.

Family History.—She has had no miscarriages and one stillbirth. She has three children, two of whom are healthy and bright and the third is subject to con-

vulsions. Her husband is living and well. No similar trouble is known in the family.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—None were noted excepting that the patient tends to hold her left hand and arm with her right hand.

Gait.—This is irregular with a left limp and produces an impression of combined paresis and ataxia. It was not very spastic but seems to have become more so and was much the kind of gait to be expected with a combination of posterior column and pyramid lesions. She walks with the left arm stiff and semi-flexed.

Coördination. — Equilibratory. When standing with feet together, both with eyes open and closed, she is rather unsteady and tends to fall to the left. There is no marked Romberg.

Non-equilibratory.—The patient is right-handed. The finger-to-nose test was performed well with the right hand. With the left hand it was performed, when first observed, with a wavering movement which rather suggested weakness of the parts involved. In subsequent examinations, it had more the character of a distinct, coarse irregular tremor. This tremor did not have quite all the characteristics of an intention tremor. It was more marked at the beginning and during the act but tended to diminish toward the very conclusion of the act. A tendency for it to appear during rest was shown by the patient's tendency to hold her left hand. At the last examination, of July 30th, it was practically an intention tremor. This movement, then, would appear to have been intermediate, in some characteristics, between a paralysis agitans tremor and an intention tremor, changing in type from one to the other.

Touching the thumb with each finger tip in succession was done slowly and rather clumsily with the left.

The heel-to-knee test was not always done well with the right extremity, showing in a slight degree the same disturbance. With the left extremity this test was per-

formed poorly, irregularly, with missing and overreaching and a tendency to increase in irregularity toward the conclusion of the act. There is thus ataxia and dysmetria of the left and, in a less degree, of the right lower extremities.

There is some adiadochokinesis of the left and possibly of the right upper extremities.

Abnormal Involuntary Movements.—These have been given above.

Reflexes.—Deep: Those of the upper extremity, excepting the triceps, are active on both sides and are more active on the left than on the right. The triceps are rather slight on both sides. The patellar reflexes are slight on the right, slight and only elicitable with difficulty on reinforcement on the left. The Achilles are absent on both sides.

Superficial: The abdominals are absent. There was a well-marked Babinski and all its modifications on the left. On the right there were the same phenomena but less marked. The last examination, of July 30th, gave an impression that the Babinski was less marked on the left than previously.

Muscle Strength.—Cooperation was not good. The left fingers showed at first weakness in flexion, extension, abduction and adduction except, apparently, adduction of the thumb. The left hand showed weakness in flexion extension, abduction and adduction, most marked in abduction. The left forearm showed quite marked weakness in flexion and supination, less marked weakness in extension and pronation. The left arm showed weakness in abduction and adduction in both the lateral and transverse planes. The left thigh showed marked weakness in flexion; in fact with the patient recumbent, the left lower extremity, with leg extended, could not be completely elevated. Abduction and adduction showed comparatively little weakness. The left leg and foot showed weakness in extension and flexion.

A reexamination of the strength of the left upper extremity seemed to show less weakness than above indicated. Another

examination of July 30th showed the left upper extremity to be normal or nearly so, there being perhaps a slight weakness of the arm. There has thus been a recovery of strength probably in this member. The legs showed little change.

Muscle Status.—No marked changes in volume and consistency were noted. The upper extremities possibly showed a slight tendency to spasm, while the lower, especially the left, seemed to show, combined with this, a slightly flaccid condition. There seemed to be, perhaps, more spasticity July 30th.

Abnormal Associated Movements.—These have not been studied, but it was noticed that the Grasset-Bychowski sign (inability to elevate one extended leg with the other already elevated) was present on the left. There was some pronation spasm of the left forearm.

GENERAL SENSORY.—Considerable difficulty was experienced in making sensory tests. Even in parts there is every reason to suppose are normal she would, after giving correct responses, "run off the track," giving incorrect ones. This peculiarity seemed rather to be a form of poor cooperation, but might possibly receive a different explanation as mentioned later.

Touch.—Acuity: This was present in both feet and legs, as well as the upper extremities, but once or twice when the foot was held the patient apparently did not know it, and there is a general blunting of sensibility in the left lower extremity. Localization was also diminished in the left foot and leg.

Discrimination.—The patient would give a series of correct replies to compass points less than 5 mm. apart on the finger tips, too many to be due to accident, and then would give incorrect answers. A later examination, however, gave better results. The big toes gave indecisive results.

Pain.—This was present and appeared to be normal on feet, legs, hands and arms. At times the patient would overreact somewhat on the lower extremities, especially the left.

Temperature.—Cool and warm stimuli were used. This was normal except some overreaction to cool.

Vibratory.—This also gave rather variable results in the lower extremities but was diminished or absent in right and left big toes, ankles and shins. It is present at the pelvis and normal in the upper extremities.

Muscle-tendon.—This showed marked diminution or absence in the left big toe and foot and probable diminution in the left leg and thigh. The right big toe showed diminution and also probably the foot, leg and thigh. The right and left fingers, hands and forearms appeared to be normal despite some variability in fingers and forearms.

Stereognosis.—This is normal. Penny, dime and nickel were well differentiated by the right and left hands and fingers.

Rough and smooth were well differentiated by both right and left finger tips.

CRANIAL NERVES. *Optic.*—Vision is normal, 20/20, R and L. The fields are normal. There was some difference of opinion about the fundi, some thinking the whole right fundus was somewhat paler than the left. The balance of opinion was against this. The pupillary reactions to light and convergence are normal. The eye movements are normal except there is a slight vertical and rotatory nystagmus on looking up, possibly a very slight on looking laterally. There is a slight von Graefe.

Facial.—The facial movements are normal. The patient smiles with the left side of the face somewhat more than with the right at times.

Hypoglossus.—The movements of the tongue are normal.

MENTAL STATUS.—Peculiarities in cooperation have already been noted. The patient impresses one as not very intelligent and a rather peculiar personality, but this appears to be more a personal idiosyncrasy than a pathologic defect.

SYSTEMIC.—The thyroid is easily palpable. The heart is normal.

The urine is normal excepting a very faint trace of albumin. The blood pressure is 115 systolic and 75 diastolic. Blood Wassermann is negative.

DISCUSSION

We have, in brief, a weakness, a diminution in deep sensation, a loss or diminution of deep reflexes and ataxia of the left lower extremity together with a Babinski; a weakness and increase of deep reflexes and abnormal involuntary movements of the left upper extremity without clearly demonstrable sensory changes. There is absence of abdominals on both sides, a diminution of deep reflexes and deep sensation (toes) in the right lower extremity. There is a history of sudden attack last May involving the left lower extremity and sudden attack of weakness with shaking involving the left upper extremity a month later and there is also a history, apparently, of some kind of third attack July 7th of a sensory nature involving the right eye, with pain. Of this last there are at present no demonstrable objective visual signs.

It will be advantageous to discuss the case first from a purely anatomical point of view. It has occurred to the writer and been advocated by a member of the staff that the lesion or lesions are all high lesions, either cortical or subcortical. In favor of a cortical lesion is: (1) The onset—a series of attacks, two at least of a monoplegic character. An extension of a hemorrhagic lesion, for example, might explain this very well. (2) The anesthesia may well be of the cortical or subcortical type affecting as it does principally deep sensation. There is one slight qualification here, however: vibratory appears to be affected as deeply as the sense of position and movement, which is probably not usually the case in cortical anesthesia, appealing, as vibration does, to thalamus as well as cortex. On the other hand the variability of results attributed to poor co-operation might very well be due to a cortical lesion, although perhaps here too widespread to be considered as such.

On the other hand there are some grave objections to attributing the symptoms to a cortical lesion or at least to a cortical lesion alone: First, there is probable absence of loss of consciousness, not decisive though with a small lesion, perhaps. Second, there is the presence of the tremor. It seems rather probable that tremors of this character are not usually due to purely cortical lesions. Third, there are the presence of deep sensory changes and also, the diminution of deep reflexes in the *right* lower extremity. This would render necessary an extension of the lesion to the other hemisphere, which is improbable unless an epidural hemorrhage is postulated, which would hardly produce the above kind of symptoms. Fourth, there is the loss or diminution of deep reflexes in the lower extremities. This latter can only be attributed to lesions of the primary deep sensory or of the lower motor neurons.

If we shift the lesion lower, to a subcortical location, we remove the above objections except the last two. The involuntary movements of the arm appear to me very like the anomalous posthemiplegic tremors or, rather, abnormal involuntary movements. They appear to have shifted from a rest movement to practically an intention tremor. There is not a pure ataxia now or at the beginning and I believe we must assume a subcortical, possibly partially lateral thalamic lesion (see below) involving the pyramid and the corpus striatum or some of its connections. The transitory character of the arm paralysis also favors such a lesion as far as the arm is concerned.

The above objections, however, of bilaterality of the leg symptoms and the loss of the lower deep reflexes, remain; and to account for these we must postulate another lesion or possibly two lesions, in the spinal cord, which would explain the first or leg attack. Such lesions involving the posterior columns and pyramids in the lumbar or low thoracic cord, and greater on the left, would probably account for the phenomena shown in the

lower extremities, i.e., the weakness, Babinski, loss of deep reflexes and ataxia, together with the rather hypotonic spasticity, if such a term be permissible. Bilateral lesions are more probable in the cord. It will be noted also that there is no history of shaking of the legs and, furthermore, the disorder of movements here is not quite of the same character now as that of the arm. A spastic ataxia would, perhaps, characterize the former. It must be remembered, too, that the spinocerebellar tracts might well be involved. The leg movements, however, may be due to further complicating factors. It is not improbable that the effects of the high (arm) lesion extended to the leg, and if this were the case we would have a movement disturbance due to a combination of pyramidal spasticity, posterior column and possibly spinocerebellar tract hypotonia and ataxia and corpus striatum disturbance. Against the presence here of the latter may be urged the absence of abnormal involuntary movements, but it is hard to tell a priori what would be the effect of a corpus striatum defect acting upon a cord already damaged as above indicated. It is a pity the leg movements could not have been examined before the second or arm attack. In the same way it is difficult to tell whether all of the sensory symptoms in the legs are due to the cord lesions. There are evidences of thalamic affective changes, but it must be remembered that there may be some cord hyperesthesia due to cord lesions alone, possibly due to the interruption of the afferent pallial paths in the cord instead of in the thalamus. This would remove the necessity of postulating a thalamic lesion, which is improbable anyway in view of the absence of sensory disturbances in the arm, and would reduce the arm lesion to a corpus striatum plus pyramid lesion.

If we grant that there have been visual disturbances of the right eye we must, it would seem, have another high lesion to account for this, also in this case on the right side. The absence of hemianopsia

appears to render the first high lesion unavailable to explain the eye symptoms. The pain, however, which occurred at the same time and the shaking would probably be due to an exacerbation of the first lesion, and it appears to me possible that the apparent visual disturbance might conceivably be due to some diffuse vasomotor or secretory effect. The patient complained of a mist or veil which she would try to rub away and apparently with some success. It is doubtful that the fundus showed changes. As mentioned below, another explanation is, perhaps, more probable.

The assumption of multiple lesions removes, then, the objections above mentioned but creates fresh difficulties. Multiple vascular lesions occurring at such widely separated points appear to be quite improbable, especially when occurring at such short intervals. The anamnesis certainly conveys the impression that all the symptoms are due to one pathologic process. Multiple sclerosis suggests itself, but even here there are objections. The onset is not characteristic of multiple sclerosis but does nevertheless occur; nor is the appearance of such profound changes as anesthesia and considerable pareses characteristic of its early stages. The tendency to remission or improvement and the possible fleeting visual disturbances, however, look very much like multiple sclerosis, which is likewise the one disease not inconsistent with multiple widely separated lesions occurring at nearly the same time and evidently all belonging to the same pathologic process. The slight nystagmus observed July 30th strengthens the case for multiple sclerosis and shows the existence of still other lesions than those discussed above.

DISCUSSION BY STAFF

DR. TILNEY emphasized the importance of first ascertaining whether we were dealing with a functional or organic condition. In the present case, however, the evidence of organic trouble was so massive that this could be regarded as settled. Cortical locali-

zation would require too widely diffused lesions, especially in view of the absence of other general signs. The abnormal involuntary movements indicated a corpus striatum lesion and the loss of deep reflexes necessitated another lesion in the cord, involving posterior columns and pyramids. He pointed out that the abnormal involuntary movements shown by the arm were not of the rhythmical tremor type but really a dyssynergia. He considered the case one of multiple sclerosis. There is no "typical" multiple sclerosis because it is multiple. The more recent tendency has been for American neurologists to diag-

nose such atypical cases as multiple sclerosis with the result that their percentage of multiple sclerosis has approximated that of European neurologists.

DR. ARONSON called attention to the importance of the nystagmus.

DR. CASAMAJOR said he thought that multiple sclerosis would be shown to be not a single entity etiologically and pointed out that besides the typical plaques there were also vascular lesions.

DR. STRONG said he did not regard the sudden onset an objection to multiple sclerosis so much as the depth of the changes for an early stage of the disease.

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THE FUNCTIONAL SIGNIFICANCE AND PRINCIPAL SYNDROME OF THE CEREBELLUM

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PART I.

A GENERAL VIEW OF THE EVOLUTIONAL SIGNIFICANCE OF THE CEREBELLUM

No part of the nervous system has been more thoroughly investigated or more the subject of debate by physiologists, anatomists and clinicians than the cerebellum. An appreciation of its functional significance must therefore depend upon a comprehensive survey of its relation to the organization of the central nervous system, to which end its comparative morphology is indispensable.

A Suprasegmental Portion of the Nervous System: The cerebellum is the expanded dorsal portion of the metencephalon. In mammals it is next in point of size to the cerebral hemispheres, from which it is separated by a membranous or bony plate, the *tentorium cerebelli*. In the lower vertebrates the cerebellum, although variable in size, is constant in its general characters. Because of its position above the segmented parts of the neuraxis it is called *suprasegmental*. This designation recognizes not only the cerebellar relation to the rest of the central nervous system but is meant even more to imply a sublimation in this organ of a certain control of special functions over which the cerebellum stands supreme. There are two other suprasegmental structures in the brain besides the cerebellum; the tectum of the midbrain, and the cerebral hemispheres. All of these suprasegmental organs, however, have the same general significance; they serve the purposes of some special functions for which the segmented portion of the neuraxis in itself does not provide; and they are, by their morphological nature, capable of great expansion where the demands of adaptation require the most complex correlation of nerve impulses. Contrasted with the suprasegmental parts of the brain, the segmental portion of the neuraxis presents a decided inflexibility in its morphological character

and is susceptible of but a small degree of expansion. It was originally endowed with and persistently retains the control of definitely fixed and fundamental organic reactions. It is the foundation without which a suprastructure would be impossible. The ground plan of the segmented portion of the central nervous system in vertebrates is constant. The decisive differences in the form of the brain occur in its suprasegmental parts, and as these latter vary in their degree of development, so animals differ in their range of adaptability to their environment.

The cerebellum, like the other suprasegmental organs of the brain, differs from the segmented portions of the neuraxis in certain essential particulars:

1. The cerebellar gray matter forms a cortex surrounding the white matter. This is a reversal of the relation of the gray to the white matter in the segmented portions of the nervous system. Such a position of the cell-containing substance greatly enhances the opportunity for expansion, since the cortex is not hampered, as is the case in the spinal cord and medulla, by a heavy investment of white matter.

2. The cerebellar connection by means of peripheral nerve fibers with receptors and effectors of the body is much less direct than that of the segmented parts. This undoubtedly provides for less immediate reflex action and also makes possible a greater degree of correlation of nerve impulses in each cerebellar reaction.

The Phyletic Constancy of the Cerebellum: The cerebellum is an organ of great antiquity. Even among invertebrates, certain arthropods (crabs), according to Bethe,⁷ have a structure in the central nervous system whose functions appear to be analogous to those of the cerebellum. Among vertebrates it is a constant element

of the brain, although it is subject to pronounced variations. The variability of its development depends upon a definite rule. Those animals which are capable of limited and simple movements possess a small and simple cerebellum, whereas animals having a wide range of motor activity possess a highly developed cerebellum. The organ appears in its simplest form in cyclostomes and amphibia, animals whose activities are relatively the most sluggish.

A brief review of the appearance of the cerebellum in the several classes of vertebrates serves to emphasize the importance of this rule.

The Cerebellum in the Different Classes of Vertebrates: In *cyclostomes* the cerebellum is but little developed; it appears as two slight dorsolateral evaginations of the metencephalon connected across the fourth ventricle by a central arched portion. The most conspicuous parts are its dorsolateral evaginations. These are in direct connection with the somatic sensory tracts and receive some general and special cutaneous nerve fibers. It is probable that the cerebellum is connected with the gustatory sense in these forms. Histologically, there is evidence of beginning differentiation into the types of cells characteristic of the cerebellum, for certain large cellular elements are present, both in the dorsolateral evaginations and in the central arch. Many medium sized nerve cells also appear and tend to take up positions in a deeper stratum. These cellular elements are doubtless the forerunners of the Purkinje cells and the cells of the granular layer. Axones from some of the large cells follow a course similar to those seen in secondary cutaneous tracts and decussate in the mesencephalon. Johnston¹⁷ believes they may represent fibers in the superior cerebellar peduncle of the higher vertebrates which arise in the dentate nucleus and reach the red nucleus in the midbrain. If such is the case these large cells themselves are the prototypes of the cellular elements later found in the dentate nucleus.

This simple form of cerebellum corresponds with the sluggish motor habits of the cyclostomes, animals which move along near the bottom of the sea or in streams.

In *selachians* (the sharks and rays) the cerebellum shows a marked advance in development; it consists of a large arch in the reduplicated roof-plate of the metencephalon, having many evaginations into which recesses from the cavity of the fourth ventricle extend. Its greater volume as compared with the cyclostomes is consequent upon the greatly increased size and complexity of its somatic sensory portion. Histologically, the organ shows much further specialization than in the cyclostomes. There are many more granular cells which form a dense, compact *granular layer* whose axones project into a more superficial zone forming the *molecular layer*. The larger cellular elements have many of the histological characters of the typical *Purkinje cells* and are arranged as a single row on the border between the molecular and granular layers. The cerebellum receives root fibers from the somatic sensory nerves and is in connection with secondary tracts from somatic sensory centers.

These connections indicate that the organ acts both as a primary receiving center of somatic sensory impulses from the muscles, skin, ear and eye, and is an area for the correlation of impulses necessary to somatic sensibility. In the more lateral portions of the cerebellar evaginations are situated secondary splanchnic sensory nuclei related to the gustatory sense. Axones from these nuclei pass inward to form a decussation in the anterior medullary velum in close relation with the decussation of the trochlear nerves. This is the *decussatio veli*.

The selachians are free-swimming fish possessed of great speed and power of movement. Their motor activities carry them to great depths in the water and enable them to swim at all levels for long distances.

In *teleosts and ganoids* the cerebellum is

not essentially different from that of the selachians. The somatic sensory portion in itself is not so large, but it has a richer connection with other centers and thus becomes more important in the correlation of sensory impulses received from the muscles, skin, ear and eye. The splanchnic sensory nuclei are much larger than in the selachians, due to the greater development of the gustatory organs in the bony fish and ganoids. The central arch of the cerebellum is larger than the lateral evaginations and is thrown into many folds.

In their motor activities, the bony fish and ganoids are similar to the selachians.

In *amphibia*, due to the generally sluggish habits of these animals, the cerebellum is small, and next to the cyclostomes, the most simple of all vertebrates. Its reduction in size as compared to selachians and the bony fish is due to the small number of special cutaneous and gustatory organs. The lateral evaginations which contain the somatic sensory elements form a simple arch over the ventricle but present no reduplication. The granular, molecular and Purkinje layers, however, are well marked. The more active amphibia, such as the frog, have much larger lateral lobes, due to an increase in the secondary tracts connected with muscular, cutaneous, vestibular and optic receptors. The cerebellum in such forms is more highly organized as a correlating mechanism.

In *reptiles*, with the exception of the crocodile, alligator and turtle, the cerebellum is small. In these more active forms it is of large size.

In *birds*, an important advance is witnessed in the enormous development of the central arch or keystone of the cerebellum, which becomes much convoluted to form the *vermis cerebelli*. This part of the cerebellum does not, properly speaking, appear in the lower classes of the vertebrate series. The lateral cerebellar evaginations are present in much reduced form as the *flocculus* and *paraflocculus*. Evidence is wanting at present to warrant a complete

statement regarding the evolution of the cerebellum in the higher vertebrates, although the conspicuous development of the vermis in birds seems to be indisputably connected with the necessities of flying. The cerebellum in ratite birds, such as the ostrich and cassowary, has a smaller vermis than in the bird of passage or in those birds whose locomotion depends mainly upon the wings.

In *mammals*, all of the elements necessary to cerebellar development in the lower vertebrates are present, including the central arch and the two lateral evaginations. The central arch becomes somewhat more complex even than in birds, but it is the expansion of the lateral evaginations in forming the hemispherical lobes that differentiates the mammalian cerebellum from all others. The *hemispheres* of the cerebellum are the last parts phylogenetically to make their appearance, although it is incorrect to maintain that the vermis appears before the lateral lobes. The earliest portions of the cerebellum are the two lateral evaginations which become connected across the ventricle by the central arch. The primitive derivatives of the lateral evaginations are the flocculus and paraflocculus. Subsequently a more cephalic area of the lateral evaginations gives rise to the hemispheres. The essential changes upon which depends the formation of the mammalian cerebellum are:

1. The increase in size and complexity of the central arch in forming the vermis.
2. The development of the lateral evaginations cephalad of the flocculus and paraflocculus to form the cerebellar hemispheres.
3. The persistence of the flocculus and paraflocculus in rudimentary conditions, these structures in the more primitive forms being the chief representatives of the lateral evaginations.
4. The submergence of portions of the lateral evaginations to form the

dentate nucleus and other medullary nuclei in the cerebellum.

The vermis, hemispheres, rudimentary flocculus and paraflocculus are the essential features of the mammalian cerebellum. These anatomical divisions of the organ vary in their development in different orders of mammals, according to the needs of the animal.

The cerebellum in the lowest mammals, the *monotremes*, has a large vermis but small and simple hemispheres. Among *marsupials*, those forms requiring much synergic control, such as the kangaroo, have large hemispheres and vermis, while the opossum, whose motor activities are limited, has a simple cerebellum.

The cerebellum is relatively simple in *rodents*, whose locomotion carries them near the ground and whose general motor capacity is not extensive. In contrast to the rodent, the *carnivores*, in response to their needs of locomotion which carries the body at a distance from the ground, have a more complex cerebellum. The great power of these animals, either in springing or climbing, makes still further demands upon the coordinating organ.

The *ungulates* have an especially well developed cerebellum, particularly those whose extremities are long, such as the camel and giraffe. In the *proboscidea*, the cerebellum attains an exceptional size. This is the case in elephants, whose great body weight, being sustained and carried at some distance from the ground, requires an extensive coordinative control to maintain equilibrium.

Certain of the *aquatic carnivores*, for example the seals, have a large and highly developed cerebellum by means of which they are enabled to execute the complex acts necessary to swimming and the performance of the swift correlated movements of the head and trunk by which they defend themselves and procure their food.

But by far the most highly developed cerebellum is seen in the *primates*. The *anthropoid apes*, representing as they do

the transition from the quadruped to the biped, possess a complex cerebellum which, in the main, is the morphological response to the needs of coordination in arboreal life, but which also foreshadows the inception of the process that has freed the upper extremities from responsibility in locomotion. This process has resulted in the establishment of a motor organ which eventually comes to have the widest range of motor activity, the human hand and arm. The relative size and complexity of the cerebellum among the different forms of primates is of interest. Those living a strictly arboreal life show a pronounced development in the vermis and the superior surfaces of the hemispheres. Those forms in which the biped tendency has made further advance with the gradual freeing of the upper extremities from direct participation in locomotion, such as the *gorilla*, *chimpanzee* and *orang-outang*, have a greater degree of development in the inferior surfaces of the hemispheres. This morphological fact is of much significance since it is in full accord with the now generally accepted conceptions of cerebellar localization. Considering the requirements of such a mobile organ as the human hand and arm and the demands of synergic control in the performance of biped locomotion, it is not surprising to find the most highly developed cerebellum in man.

Generalized Pattern of the Cerebellum in Mammals: Descriptions of the cerebellum have caused much confusion because they have been based upon the conditions of the most complex of all forms, man. Elliot Smith³⁰ and Bolk⁸ have demonstrated that such divisions as have been employed for the human cerebellum will not meet the requirements in the different orders of mammals. A simplified scheme for the divisions of this organ has been devised by Elliot Smith and supplemented in a later work by Bolk. Smith's studies were based upon the cerebellum in the edentata; those of Bolk were made upon the cere-

bellum of lemur albifrons. Both investigators recognize an *anterior* and a *posterior cerebellar lobe* separated from each other by a large fissure which appears early in development, the *fissura prima*. The posterior lobe is further subdivided by a second fissure appearing later in development, the *fissura secunda*. As growth proceeds, the anterior lobe becomes subdivided by other secondary fissures, i. e., (1) the *fissura preculminata* and (2) the *fissura lingualis*.

These fissures determine three subdivisions in the anterior lobe, the most cephalic of which is the *lingula*, the middle, the *pars preculminata*, and the most caudal, the *pars culminis*. The posterior lobe shows much more subdivision. In it are recognized a median portion, corresponding in position to the vermis, which is divided into four parts by three parallel fissures, (1) the *fissura suprapyramidalis*, (2) the *fissura secunda* and (3) the *fissura postnodularis*. These fissures divide the median portion into the *pars suprapyramidalis*, the *pyramid*, the *uvula* and the *nodula*, all being more or less distinct divisions of the vermis. The lateral expansion connected with this portion of the vermis is divisible into two parts, the *pars lateralis* and the *pars flocculi*. The *pars lateralis* is the lateral expansion of all of that part of the vermis cephalad of the *fissura secunda*, while the *pars flocculi* is the lateral expansion of the nodule and uvula. The *pars lateralis* is subdivided into certain smaller areas by the appearance of three radiating fissures which converge upon the vermis. These fissures are the *fissura postlunata*, the *fissura postpteroidea* and the *fissura parapyramidalis*. (Fig. 1.)

The area cephalad of the *fissura postlunata* is the *area lunata*. Caudal to this in regular succession are the *area pteroidea*, the *area postpteroidea* and the *area parapyramidalis*.

The *pars flocculi* is divided by a fissure which extends inward from the lateral extremity about one-third of the distance toward the nodule; this is the *fissura*

floccula, which separates the *paraflocculus* from the *flocculus*. The *fissura parafloccula* intervenes between the *pars lateralis* and *pars flocculi*.



FIG. 1. SCHEMATIZATION OF THE FUNDAMENTAL ARRANGEMENT OF THE PARTS OF THE MAMMALIAN CEREBELLUM SPREAD OUT IN ONE PLANE, ACCORDING TO ELLIOT SMITH AS A RESULT OF HIS ORIGINAL WORK ON THE CEREBELLUM OF THE EDENTATA.

This pattern, according to Elliot Smith, may be applied to the analysis of the cerebellum of all vertebrates with the possible exception of monotremes.

Bolk, as the result of a study of the cerebellum in a large comparative series, still further advanced the ideas put forward by Elliot Smith. He concluded that the several parts recognizable in the mammalian cerebellum have definite functional significance and represent areas of central control over definite motor performances in the body. According to Bolk, the cerebellum is divided into an anterior and posterior lobe by the *sulcus primarius* which corresponds to the *fissura prima* of Smith. (Fig. 2.) The anterior lobe is divided by a series of three fissures into four lobules which Bolk numbers 1, 2, 3, and 4. A small and shallow fissure parallel to the *sulcus primarius* delimits the *lobulus simplex* which extends from the vermis out upon the lateral expansion of the hemispheres. The major portion of the posterior lobe, however, that which lies caudal to the *lobulus simplex*, is divided into a median lobule and two lateral lobules. The median lobule constitutes the vermis and is separated from the lateral lobules by the two sulci

paramediani, one of which lies on either side of the vermis. The vermis itself is divided into four lobules, respectively indicated as C-2 and C-1, B and A. The fissura secunda passes between C-1 and B. The lateral lobules are divided into two main

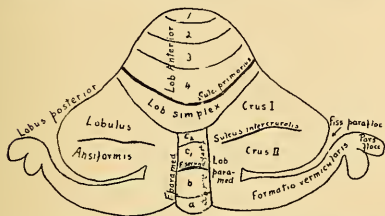


FIG. 2. SCHEMATIZATION OF THE FUNDAMENTAL ARRANGEMENT OF THE PARTS OF THE MAMMALIAN CEREBELLUM SPREAD OUT IN ONE PLANE, ACCORDING TO BOLK, WHO SELECTED FOR HIS TYPE THE CEREBELLUM OF LEMUR ALBIFRONS.

portions, the *lobulus ansiformis* and the *formatio vermicularis*. These are separated by a deep incisure, the largest fissure in the cerebellum, the *fissura parafloccula*. The lobulus ansiformis is divided by a transverse fissure, the *sulcus intercruralis*, into Crus I and Crus II. Where the lobulus ansiformis and the formatio vermicularis come into relation with the posterior median lobule, there is a portion of the cerebellum which Bolk defines as the *lobulus paramedianus*. He explains the striking variations of the cerebellum on the grounds that form is dependent upon function and there must, therefore, be some direct functional relationship between these variations in the cerebellum and the control of the muscular system whose tonic, sthenic and static activities are dependent upon this organ. He concluded that all of the muscles of the body may be divided into two groups: those which act bilaterally together such as the trunk muscles, and those which, although bilaterally present, have a certain independence in their activity like the muscles of the arms and legs. The muscle groups which show this independence of action are unilaterally synergic, while those which depend upon a

simultaneous coordination are bilaterally synergic. To the latter group belong the muscles which produce movements of the head, of the eyes, of the mouth and jaws, of the tongue, pharynx and larynx, and of the trunk, in addition to certain movements of the upper and lower extremities which require bilateral innervation and act together as in locomotion. Upon this hypothesis, Bolk based his localization in the cerebellum of control for the two major groups of muscles. The cerebellar cortex is composed in mammals of a number of coordinating centers, some of which are paired for bilateral synergic control, while some are unpaired for unilateral synergic control. (Fig. 3.)

The *lobulus anterior cerebelli* contains the coordinating centers for the muscles which are active in movements of the head. The most anterior lobule is for the eyes; the second lobule, for the tongue; the third, for the muscles of mastication, and the fourth, for the muscles of expression and of the larynx and pharynx. The *lobulus simplex* contains the paired synergic centers for the muscles of the neck. In the upper portion of the lobulus medianus posterior are the paired centers for movements of the right and left extremities. The lobuli ansiformes and paramediani contain unpaired centers for the extremities, the arrangement of the centers in each case being ipsilateral, Crus I corresponding to the arm of the same side and Crus II to the leg of the same side. The remainder of the cerebellum contains coordinating centers for the trunk and the tail region, the formatio vermicularis particularly having control of the tail.

This localization in the cerebellum by Bolk on the grounds of comparative anatomy was subsequently confirmed by André-Thomas and Durupt² and by Van Rynberk³³ through many experiments upon animals.

Uniformity of the Internal Structure of the Cerebellum: Throughout the vertebrate series, the cerebellum presents a

the roof of the fourth ventricle mesial to the nuclei globosi. These are the *nuclei tecti* or *fastigii*, while somewhat ventro-mesial to the nucleus dentatus in mammals is a large collection of nerve cells, the *nucleus emboliformis*. Collectively, these nuclei occupying positions in the white matter are spoken of as the *medullary nuclei* of the cerebellum.

Even within the narrow limits of the primates, these nuclei vary much in their distinctness and prominence. It is only in man, the gorilla, chimpanzee, orang-outang and the gibbon, that the nucleus dentatus has the typical convoluted appearance which resembles the inferior olive of the medulla, a fact which has led to the term, *oliva cerebelli*. In the lower apes, the convoluted appearance of the cerebellar olive is entirely wanting.

Primitive Connections of the Cerebellum:

The cerebellum receives root fibers from several somatic sensory nerves, notably the fifth, which, in the fish, establish a primary connection with lateral line organs of the supraorbital, infraorbital and hyomandibular rows. In many of the fish, there is a large vagus connection probably from the lateral line organs of the body. In all of the lower vertebrates, including birds, the cerebellum is connected with secondary somatic sensory tracts of the somesthetic, optic and auditory types, which latter tract in all probability is vestibular. There is a primary splanchnic sensory connection in the cerebellum serving the gustatory sense and likewise a secondary pathway through the cerebellum for this type of sensibility. All of the more exact connections of the cerebellum among vertebrates are difficult to enumerate at the present time. The vertebrates, exclusive of birds and mammals, possess two tracts which connect the diencephalon with the cerebellum. These are particularly well developed in teleosts and selachians, although less well defined in amphibians and reptiles. They are the *tractus diencephalocerebellares*. In fish, amphibia and

reptiles, the brachium conjunctum arterius contains three main tracts:

1. *The tractus tegmento-cerebellaris* which arises in the ganglion at the caudal end of the tegmental portion of the diencephalon and is a crossed connection with its decussation in the cephalic extremity of the midbrain.

2. *The tractus mesencephalo-cerebellaris* which is also a crossed connection with its decussation in the superior medullary velum in close relation with the decussation of the trochlear nerve.

3. *The tractus tecto-cerebellaris* which is likewise a crossed connection arising in the tectum of the mesencephalon with its decussation in the velum, forming part of the *decussatio veli*.

In all vertebrates, the brachium posterius, known in mammals as the inferior cerebellar peduncle, contains fibers from the following sources:

1. *Tractus spino-cerebellaris*, both a direct and crossed connection.

2. *Fibræ arcuatae anteriores and posteriores*.

3. *Tractus vestibulo-cerebellaris* and, in selachians and teleosts, a *tractus trigemino-vago-cerebellaris*.

In mammals, the posterior brachium is augmented by a large contingent from the inferior olive constituting the *tractus olivo-cerebellaris*.

From these connections it is evident that the cerebellum even from its earliest history has borne an extensive relation to all the different types of sensibility, including somesthetic, optic, auditory and gustatory types. It is also provided with an ample association system which permits the organ to serve as a center of correlation. This sensory correlation has had, from the inception of cerebellar development, a definite purpose, as shown by the internal structure of the cerebellum which has maintained a constant morphological character. The function of the organ must, therefore, have been constant and its correlation of sensory impulses derived

from so many different sources must have had a common aim. All the facts seem to indicate that this correlation was in the interest of regulating motion by the addition to motor performance of that essential element termed synergic control.

The Connections of the Cerebellum in Mammals: With the exception of the gustatory connection which no longer plays a part in the sensory contributions entering into cerebellar correlation, the sensory tracts to the cerebellum in mammals indicate that the essential nature of the organ has remained unchanged through the later stages of evolution. Three peduncles with certain accessory connections serve to connect the mammalian cerebellum with the rest of the central axis. These are the *superior cerebellar peduncle* corresponding to the brachium conjunctivum anterius, the *inferior cerebellar peduncle* or *restiform body* corresponding to the brachium posterius, and the *middle cerebellar peduncle*, a mammalian acquisition not present in the lower vertebrates. According to the observations of Van Gehuchten³² the following functional groups of fibers participate in these cerebellar connections:

1. Through the inferior cerebellar peduncle which contains:
 - a. The dorsal spino-cerebellar tract from the muscles, joints and tendons.
 - b. A secondary connection with the columns of Goll and Burdach through the anterior and posterior arcuate fibers establishing direct and crossed connections, also from the muscles, joints and tendons; possibly from the skin.
 - c. The olivo-cerebellar tract, direct and crossed, connecting both inferior olives with the cerebellum, coordinating the movements of the head with those of the eyes.
2. Through the juxtarestiform body which contains:

- a. A Deitero-cerebellar tract connecting the nucleus of Deiter with the nucleus tecti of the cerebellum for equilibratory control.
 - b. A secondary connection between the substantia gelatinosa, representing the trigeminal nerve, and the vermis of the cerebellum.
3. Through the superior cerebellar peduncle which contains:
 - a. The dentato-mesencephalic and the dentato-diencephalic fibers, the former ending in the red nucleus after a complete decussation in the midbrain, and the latter extending to nuclei of the thalamus. It is possible that some fibers extend from the red nucleus to the dentate nucleus, but the superior cerebellar peduncle serves as the chief pathway for impulses leaving the cerebellum.
 4. Through the middle cerebellar peduncle which contains fibers serving as a connection between the frontal, occipital, parietal and temporal lobes of the cerebral hemispheres and the lateral lobes of the cerebellum. This establishes a completely crossed connection through the pontile decussation.
 5. Through the fibers of the ventral spino-cerebellar tract which enter the vermis of the cerebellum after traversing the lateral aspect of the superior cerebellar peduncle.

It is evident that the cerebellar connections in mammals indicate the same functional capacity as in the lower vertebrates. The fibers in the inferior cerebellar peduncle represent an extensive connection between the muscles, bones and joints and, perhaps to some degree, the cutaneous surfaces of the body. The juxtarestiform body is indicative of a connection between the semicircular canals, utricle and saccule of the internal ear on

the one hand and the cerebellum on the other, still retaining the ancient relation which, in many of the lower vertebrates, is much more extensive because it includes the organs of the lateral lines. The connections through the middle cerebellar peduncle provide opportunity for more efficient sensory associations than in the primitive forms, since they mediate sensory activities arising in the occipital, temporal, parietal and frontal lobes of the cerebral hemispheres and serve respectively for the ultimate elaboration of visual, auditory, somesthetic and kinesthetic impressions.

The superior cerebellar peduncles represent amplified pathways by means of which fibers leave the cerebellum for the spinal cord and brain-stem. This is a more ample connection, because the correlation which occurs in the cerebellum of mammals, being more extensive and complex, needs a larger pathway for distribution.

The Evolutionary Significance of the Cerebellum: From the early stages of vertebrate history, the cerebellum has served as an organ of association for sensory impulses which are here correlated and distributed in order to maintain the muscles of the body in a state of coordination. The advantages accruing to this process from the combination of vestibular, auditory, somesthetic and optic impressions cannot be called in question.

The most primitive vertebrate motion depends upon the harmonious innervation of the axial musculature, whose action must be coordinated from body-segment to body-segment and synchronized upon the two sides of the body. Such animals as depend upon swimming movements for their locomotion and in which swimming movements represent the maximum motor achievement, require a bilateral synergic control. The mechanism best calculated to supply this control would consist of paired interdependent collections of coordinating cells. Such an organ is the cerebellum in all of the fish. In some lower

vertebrates the necessity for this bilateral synergic control is much less in demand than in others, as for example the cyclostomes, which are animals solely dependent upon a vermicular movement for all their motor activity. A similar vermicular movement is sufficient in the reptiles to carry them over the surface of the ground. This is also true of the more sluggish of the amphibia whose movements are of a vermicular character. Even the more active amphibians such as the frog are fundamentally dependent upon vermicular movement. In their larval state, locomotion resembles that of other swimming animals. Their extremities when acquired have no great independence one from the other, but are regulated in such a way as to cooperate almost as a single organ. In fact while certain of the amphibia do possess extremities, these appendages are bilaterally synergic in their action. In the same manner the movement of the birds, both those depending upon the wing and the running bird, require bilateral synergic control. This is especially true in flight where the innervation of the wings must be subjected to the most exact sort of bilateral control. In the mammal a new phase of motion is developed with the appearance of extremities, each of which has actual independence of action. An increasing degree of such independence determined the semibiped condition, then the biped, and finally released the upper extremities from responsibility in locomotion. But even before this independence of the arms was achieved in the biped, many of the quadrupeds gained the ability to control one foreleg independently of the other and had a similar independence in the hind extremities. It was largely in consequence of these unilaterally synergic activities that the hemispheres of the cerebellum made their appearance. This fact becomes significant in the light of Bolk's investigations which go to show that in all mammals the lobulus ansiformis in its two divisions, the anterior and posterior crura, is present, and furthermore, that

these parts become increasingly more complex with the increased complexity in the independent movements of each limb.

In summarizing the evolutional significance of the cerebellum, it may be concluded:

1. That the cerebellum has been added to the neuraxis as a suprasegmental structure in the interest of coordination.

2. That in its primitive form it correlates sensory impulses received from all of the chief receptors of the body and utilizes the impulses thus associated most advantageously to accomplish the purposes of coordination.

3. That in the lower vertebrates this coordination requires the harmonious action of the axial musculature and depends upon the bilaterally synergic control of the central arched portion of the cerebellum, corresponding in position to the vermis, although certain lateral structures such as the parafocculus and flocculus were added for the more or less independent movements of the tail.

4. That when the upper and lower extremities made their appearance and became capable of independent move-

ment, they needed something more than bilaterally synergic control. Out of this necessity grew the expansive lateral portions of the cerebellum, the cerebellar hemispheres, which provide unilateral synergic control for the arms and legs, exercising regulation over such independent and individual movements as these extremities have the capacity to develop.

5. That, as a natural consequence, the greater the range of independent movement in the extremities, the greater has become the cerebellar area required for their control.

6. That the greatest range of independent movement is seen in the skilled performances which are the special attributes of man, who, as a result, has the most highly developed cerebellar hemispheres.

7. That the central or vermal portion of the cerebellum still retains its primitive control not only of the bilaterally synergic movements of the eyes, face, tongue, jaws, larynx, neck and trunk, but also maintains a similar relation to movements of the extremities which require bilaterally synergic control.

PART II.

THE FUNCTIONS OF THE CEREBELLUM

Experimental Evidence: Cerebellar functions have been matters of discussion for many years. Numerous investigators have devoted their attention to this problem and the most important of their opinions concerning it are essential to an adequate conception of the organ. In the main, there is a surprising unanimity in these opinions which, although varying much in detail, have a common key-note with regard to the functions of the cerebellum.

The earliest authoritative investigations are usually attributed to Flourens,¹³ who reported his notable experimental work on birds and mammals in 1842. His observations led him to believe that the regulation of locomotor acts in walking, running, leaping and flying depends upon the cerebellum. Cuvier⁹ formulated this conception in the statement that "the integrity of the cerebellum is necessary to the proper regulation of locomotor movements."

Magendie,²¹ who repeated the original experiments of Pourfur de Petit²⁵ by cutting the cerebellar peduncles, concluded that the cerebellum is primarily the organ for equilibrium rather than for the co-ordination of locomotion, while Lussana²⁰ maintained that it is the center for muscular sense.

The conclusions of Flourens and Magendie found support in many subsequent investigations, especially by Serres,²⁹ Hertwig,¹⁶ Foville¹⁴ and Desmoullin.¹⁰

Vulpian³⁵ and Philippeaux²⁶ observed that destruction of the cerebellar connections in the fish produced disturbances similar to those described by Flourens in birds, and Goltz¹⁵ found that injuries to the cerebellum in frogs led to inexact motor control.

Weir Mitchell²³ in his studies upon birds contributed a new observation to the record of experimental work upon the cerebellum. He noted that fatigue was a con-

stant phenomenon following experimental operations and concluded that the cerebellum was, among other things, the source of energy and muscular strength.

Ferrier¹² considered the cerebellum an organ having to do with the maintenance of equilibrium, and this also was Stefani's³¹ interpretation. Nothnagel²⁴ recognized certain differences in function between the vermis and hemispheres, since it was destruction of the former only that led to motor disturbances. The hemispheres were not concerned in the control of locomotion.

To Luciani¹⁹ we are indebted for an important contribution in which he critically reviewed the previous works upon the cerebellum. Upon the basis of his own investigations on apes and dogs, he constructed the hypothesis that the cerebellum was an organ which provided a sthenic, tonic and static control for the muscles and that defects in this control led to what he termed *asthenia*, *atonia* and *astasia*, the clinical manifestation of which latter is *ataxia*. These three major symptoms are known as the *triad of Luciani* and have been generally accepted in connection with cerebellar disease.

André-Thomas¹ demonstrated the important fact that in experimental animals the symptoms were not all of the same character but depended much upon the pathological factors which produced them. Thus he noticed symptoms of irritation due to stimulation by hemorrhage or the trauma of the operation or perhaps to compression of a blood clot consequent upon the operative procedure. The second type of symptoms due to cerebellar experimental work was caused by destruction that gave rise to inhibitory defects, while the third class included changes due to substitution and compensation which made their appearance after the lapse of considerable time following the operative disturbance.

André-Thomas gave much attention to the peculiar symptoms known as *forced movements* which appear in experiment animals as a result of partial destruction of the cerebellum. He called attention to the direction which these forced movements invariably take, especially after removal of one hemisphere. A dog operated upon in this manner, after recovering from the effects of the anesthetic, tends to roll over and over, rotating about his long axis in a direction always away from the sound side and toward the side of the operation. It was also shown that animals upon which the various cerebellar operations had been performed, although they were unable to maintain an upright position in standing or walking, if placed in water, could swim. This remarkable and contradictory condition was explained by the fact that the body under these circumstances was surrounded by a medium more tangible than the air which, because of its denser nature, augmented the afferent impulses from the skin muscles and joints, thus permitting the animal, while in the water, to compensate for much of its loss in muscle and cutaneous sensory impressions. André-Thomas does not wholly agree with Luciani as to the asthenia, atasia and atonia resulting from cerebellar lesions, but believes that the organ has most to do with equilibrium.

Versilov³⁴ as a result of his experiments, concluded that the cerebellum is an organ of equilibrium, that experimental defects in equilibrium depend upon asthenia and ataxia, that disturbances apparent in the tremor and pendular movements of the head and body are increased by attempts at volitional control, that removal of the cerebellum causes increase in the deep reflexes and that the cerebellar control is ipsilateral in the hemisphere and bilateral in the vermis.

Cerebellar Localization: The first extensive attempts at cerebellar localization were made by Bolk upon a large series of the lower animals. His method of approach

to the problem, together with his conclusions, have already been summarized. (See page 294.)

Experimental investigations to test Bolk's cerebellar localization were made by Van Rynberk³⁵ working in Luciani's laboratory. Van Rynberk explored the cerebellar cortex by means of circumscribed extirpations of certain of the lobules. The results of his work confirmed Bolk's hypothesis and showed that the localization as outlined by that investigator was essentially correct. Van Rynberk's conclusions are as follows:

1. Total or partial extirpation of the lobus simplex produces side to side oscillations of the head due to an asynergia in the muscles of the neck.

2. Complete extirpation of the crus primum produces bilateral disturbances in the fore limbs which, in the first stages, appear as irritative symptoms with the assumption of characteristic positions likened by the author to the *military salute*. Later there occurred considerable asynergia in the movements of the limbs.

3. Extirpation of the crus secundum produces asynergia and asthenia of the muscles of the hind legs.

4. Extirpation of both crura of the lobulus ansiformis produces marked asynergia and asthenia in both fore and hind legs on the same side as the lesion.

5. Extirpation of the lobulus paramedianus causes rotation about the longitudinal axis of the body associated with the tendency to fall toward the operated side.

The symptoms in all cases gradually become less marked and, ultimately, almost entirely disappear owing, according to Luciani, to organic compensation.

The observations of Van Rynberk have subsequently been confirmed in all their essential details by many investigators working upon dogs, monkeys and other animals. This is particularly true of the work of Rothmann³⁶, and of André-Thomas and Durupt.² All of these investigators point with emphasis to the fact that de-

struction of the cerebellar cortex does not give rise to paralysis but does produce a disturbance in coordination of the muscles of the body.

Their investigations have carried cerebellar localization a step further by showing that within the paired centers for the upper and lower extremities there are definite subsidiary centers for the direction of antagonistic muscles. Each subsidiary center for the extremities is related to a single segment or articulation of a limb and controls the elaboration of the sthenic, tonic and static impulses distributed to a definite muscle group. An almost uniform observation as a result of experimental work upon the cerebellum is the gradual regression of the cerebellar symptoms in the course of time. This has been attributed either to an organic compensation by the uninjured parts of the cerebellum itself or to an actual functional compensation by the volitional control of the cerebral cortex.

Experimental Evidence from Stimulation of the Cerebral Cortex: The excitability of the cerebellar cortex to experimental stimulation has been the subject of much dispute. Versilov made extensive studies upon the cerebellum by means of different types of stimuli, using electrical, mechanical, and thermal stimuli. His conclusions are as follows:

1. All the phenomena which result from stimulation of the cerebellum show that its function is related exclusively to the motor sphere.

2. The motor responses of the muscles of the neck, the trunk and the extremities resemble in their general character the responses obtained as the result of stimulation of the cerebral cortex, being both tonic and clonic contractions of isolated muscle groups.

3. The relations of the cerebellum to movements of the body, of the neck, of the head and of the extremities appear to be territorial, so that the right hemisphere of the cerebellum controls the right body

half and the left cerebellar hemisphere the left body half. The middle lobe likewise has a similar partition corresponding to the right and left halves of the body. The superior vermis stands in relation to the upper extremities, while the inferior vermis controls the lower extremities.

4. In addition to its relation to the muscles of the extremities and the trunk, the cerebellum has some significance in the innervation of the cutaneous muscles.

5. The movements of the eyeball are under the control of the cerebellum. Particular areas of the cerebellar cortex when stimulated give rise to several varieties of eye movements.

6. All of the eye movements appear to be synergized.

7. Nystagmic movements of the eyeballs form an almost constant phenomenon resulting from stimulation of the cerebellar cortex. The direction of this movement is variable, yet four chief movements may be differentiated, namely, horizontal, vertical, diagonal and rotatory.

8. Besides the usual movements of the eye as a result of stimulation of the cerebellar cortex, there are movements which result in protrusion and retraction of the eyeballs.

9. Stimulation of the cerebellar cortex also causes changes in the movements of the eyelids.

Prus²⁷ likewise conducted experiments upon the cerebellum by means of various types of stimulation; his conclusions are summarized as follows:

1. The cortex of the cerebellum is excitable to electrical stimuli.

2. Excitability manifests itself in the form of tonic and clonic contractions in the muscles of the same side of the body.

3. Excitability of the cerebellar cortex is much influenced by the direction of the electric current. The longitudinal application of the electrodes in stimulating the neighborhood of the midline of the vermis brings about bilateral contractions.

4. Transverse stimulation brings out

tonic contraction while longitudinal stimulation produces clonic contraction.

5. In the vermis, there are motor centers for the muscles of the body, of the eye and of the neck. In the anterior portion of the cerebellar hemispheres are centers for the fore limbs, and in the posterior portion centers for the hind limbs.

6. The dependence of the stimulation phenomenon upon the direction of the current is due to the arrangement of the Purkinje cells, the dendrites of which extend transversely to the long axis of the folium.

Bechterew⁶ as a result of electrical stimulation of the cerebellar cortex arrived at the following conclusions:

Stimulation Confined to the Vermis:

1. Stimulation of the left side of the pyramis at its cephalic extremity produces movements of the anterior extremity on the corresponding side.

2. Stimulation of the pyramis near the spinal cord results in movements of the posterior extremity on the corresponding side.

3. Stimulation of the entire left half of the pyramis occasions simultaneous movements of the anterior and posterior extremities on the same side.

4. Stimulation of the right side of the pyramis near its middle produces movements of the right anterior extremity.

5. Stimulation of the right side of the pyramis near the spinal cord produces movements of the right posterior extremity.

6. Stimulation of the entire right half of the pyramis produces movements of the anterior and posterior extremities of the corresponding side.

7. Stimulation of the cephalic portion of the pyramis near the midline produces movements of both anterior extremities.

8. Stimulation of the pyramis in the midline near the spinal cord produces movements in both legs.

9. Stimulation of the entire pyramis in the midline gives rise to simultaneous movements of the anterior and posterior extremities.

Simultaneous Stimulation of the Anterior Portion of the Clivus and the Posterior Portion of the Culmen:

1. Stimulation of the left anterior portion of the clivus and of the sulcus which separates it from the culmen occasions movements of the left shoulder girdle and left anterior extremity.

2. Right-sided stimulation of the most anterior portion of the clivus and the fissure separating it from the culmen occasions movements of the right shoulder girdle and right anterior extremity.

3. Stimulation of the median part of the anterior portion of the clivus and its fissure occasions movements of the entire shoulder girdle together with movements of both superior extremities.

Stimulation of One-Half of the Clivus and the Culmen:

1. Stimulation of the indicated portion of the left half of the culmen and the clivus brought out during the passage of the current a deviation of the eyes and of the head toward the left. Increase of the stimulation caused a movement of the eyes to the right followed also by a movement of the head in the same direction.

2. Stimulation of the indicated portion of the right side of the clivus and culmen caused a deviation of the eyes and the head to the right; after an increase of the current the eyes and head were turned to the left.

The evidence derived from stimulation of the cerebellar cortex at the hands of several investigators seems to be conclusive not only as to its experimental excitability but also with reference to the presence of localized functional areas within it.

Clinical Experimentation and Clinical Evidence: Pathological conditions of the cerebellum have furnished useful data concerning the functions of the organ, especially those due to circumscribed lesions such as tumor, abscess or cyst. Rothmann, André-Thomas and Durupt, Babinski and Tournay⁴, have contributed by this means to the theory of cortical localization in the human cerebellum which more recently has

been elaborated by the clinico-pathological and experimental work on human subjects by Robert Barany.⁵ As the result of a large series of observations, Barany has evolved certain clinical tests by means of which it is possible to localize circumscribed cerebellar lesions with a marked degree of accuracy. One of the tests is especially pertinent in clinical localization.

Barany's Index or Pointing Test: During this test the patient's eyes are blindfolded. With his elbow on a firm support he is instructed to touch with his index finger an object held in front of him. This he does repeatedly, first in several vertical planes, then in horizontal planes. If there is a constant tendency toward deviation from the plane of movement, its direction is noted. By a slight modification of the procedure, it is possible to test each of the limb segments in all positions of rotation, pronation and supination.

These tests are performed in two ways, first, *without* previously induced nystagmus either by rotation or by the method of introducing water into the external ear; second, *after* inducing artificial nystagmus by the means mentioned. The normal subject presents the following reactions:

1. Without induced nystagmus the tests in question are correctly performed, there being no deviation.

2. After artificially induced nystagmus, a deviation is noted in the direction corresponding to the slow movement of the nystagmus. This is termed by Barany the "reaction deviation."

If there be a cerebellar lesion present, certain variations of the above reactions will be observed:

First, when tested without induced nystagmus, a definite deviation will be occasionally noted. This is what Barany termed "spontaneous deviation."

Second, after inducing artificial nystagmus, there is an absence of the normal deviation reaction.

This test has proved the most useful in diagnosis. The deviation which is observed during these tests serves to indicate the

side of the cerebellar lesion. Barany, employing this means of investigation, which he supplemented by much clinico-pathological material and experimental inhibition of the cerebellar cortex in patients during operation by rapid artificial cooling, has identified definite areas with the special muscular actions. His conclusions are summarized as follows:

1. There exists an exact localization of function in the cortex of the cerebellum.

2. The centers for the extremities are situated in circumscribed areas of the cortex of the hemispheres.

3. These centers correspond to those postulated by Bolk and experimental researches of other authorities.

4. The centers for the right and left extremities are situated respectively in the right and left superior and inferior semilunar and digastric lobules.

5. Within these centers the representation of the limb musculature is spatially determined by the action of its various functional groups and by their position in the limbs. Thus, within the arm center, other subsidiary centers are recognized governing movements of the limb in the horizontal plane, in the sagittal plane, in rotation, pronation and supination. In each subsidiary center the more minute arrangement of functional areas is in accordance with the movable segments of the limbs.

6. The sudden destruction of a center in the cerebellar cortex concerned in movements of the limb in a given direction, as for example to the right, results in the production of spontaneous deviation in the inverse direction, that is, to the left. This spontaneous deviation disappears in time, either through compensatory action of the cerebral cortex or of the cerebellum itself.

7. The principal centers of direction which have been determined are localized as follows:

- ⓐ. A center for the control of the musculature concerned in the movements of the right arm downward is situated in the median portion of

the right inferior and superior semilunar lobules.

- X. A center for the control of the muscles concerned in adduction of the right arm is situated in the lateral angle of the hemisphere in the right superior and inferior semilunar lobules. (Fig. 4.)
- O. A center for the control of the musculature concerned in adduction of the right hand is situated in the anterior part of the right digastric lobule.
- + . A center for the control of the musculature adducting the right arm is situated caudad and laterad from the preceding center in the right digastric lobule.
- + Still more laterad and caudad on the right side is situated a center for the control of the musculature adducting the right thigh. (Fig. 5.)

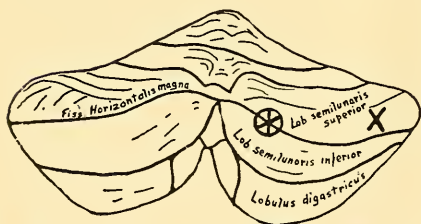


FIG. 4. SCHEMATIZATION OF THE POSTERIOR ASPECT OF THE HUMAN CEREBELLUM INDICATING CERTAIN CEREBELLAR LOCALIZATIONS ACCORDING TO BARANY.

The left hemisphere contains a similar arrangement of centers for the extremities.

The clinical evidence thus far adduced seems to be in accord with the theory of cerebellar localization originally formulated by Bolk. Barany's localizations in the human cerebellar cortex have yet to be confirmed in detail but the importance of his work cannot be overestimated.

Synergia and Synergic Movements: The recognition of the cerebellum as an organ essential to motor control is the principal feature in the conclusions of all observers. Babinski¹ expresses this consensus of opinion when he says that "every form of active muscular exertion necessitates the simultaneous cooperation of immense assemblies of synergic movements throughout the body to secure steadiness and maintain general equilibrium, and on the hypothesis that the cerebellum is the center of these unconscious adjustments, we would expect the cerebellum to be developed in proportion to the variety and complexity of the motor activities of which the animal is capable. The facts of comparative anatomy and development are entirely in harmony with this hypothesis."

The explanation of cerebellar disturbances given in Luciani's triad of asthenia, atonia and ataxia, although affording a

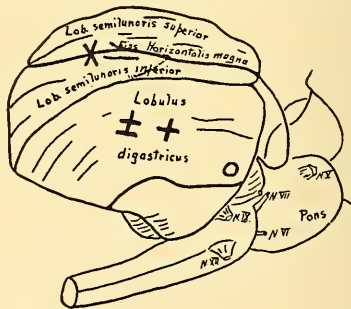


FIG. 5. SCHEMATIZATION OF THE INFEROLATERAL ASPECT OF THE HUMAN CEREBELLUM INDICATING CERTAIN CEREBELLAR LOCALIZATIONS ACCORDING TO BARANY.

N. V, NERVUS TRIGEMINUS; N. VI, NERVUS ABDUCENS; N. VII, NERVUS FACIALIS; N. IX, NERVUS GLOSSOPHARYNGEUS; N. XII, NERVUS HYPOGLOSSUS.

generalized conception of the nature of these defects, is scarcely analytical enough to meet the demands of clinical experience. Mills and Weisenburg²² in consequence have maintained that Luciani's triad requires a new interpretation. According to their view, *synergia is the fundamental*

function of the cerebellum. Their clinical studies convince them that asynergia is the fundamental symptom of cerebellar disease. Certain symptomatic modifications of this disturbance appear as dysmetria (improper measurement of movements), adiadochokinesis (inability to perform succession movements) and tremor. Asthenia and atonia are secondary effects of asynergia.

Definition of Synergia: Synergia, according to Mills and Weisenburg, is the power or faculty by which movements more or less complex but functionally definite, are associated in special acts. In a broad sense it is a motor association, this association being carried out in movements of different parts of the same limb or in synchronous movements of the limbs, of the limbs and the head, or of the trunk, limbs and head together.

The Cerebellar Gait—Trunkal Asynergia: Mills and Weisenburg, in addition to their clinico-pathological studies, employed the cinematograph to investigate cerebellar symptomatology. By this means they were enabled to study the gait in patients suffering from cerebellar diseases, the disturbances of which in the majority of cases were chiefly trunkal. The trunk may be carried either backward or forward, forward and to one side, or simply to one side alone. This inclination may be total or partial. Every attempt on the part of the patient to walk causes the trunk to lean backward, forward or to one side. It is impossible for him to stand for any length of time in the erect position. He may be able for a short time to stand or walk either with the trunk erect or leaning in some direction, but he is soon likely to lurch forward, backward or to one side. In such cases, asynergia of the trunk is predominant and forms the most impressive part of the disturbance. The legs seem to act as props; the trunk leads and whichever way it goes the legs follow. If the trunk leans forward and to the left, the swing of the left leg is shorter than that of the right, or if the trunk goes forward

and to the right, the swing of the right leg is less than that of the left and the legs finally accommodate themselves to the movements of the trunk.

Asynergia in the Pelvic and Shoulder Girdle Muscles: The observations of Mills and Weisenburg concerning the muscles of the pelvic and shoulder girdles in locomotion are especially instructive. In certain cases the asynergia is just as marked in the shoulder as in the pelvic girdle. When the asynergia is most marked in the pelvic girdle, the legs are held farther apart, the arms nearly always are outstretched and semiflexed at the elbows. When the asynergia is shown in the pelvic movements alone, the trunk is held rigidly erect and the legs are moved forward from the pelvis, giving the impression that the trunk follows the legs. In the extreme pelvic girdle cases, it is difficult for the patient to walk without assistance, and when assisted, the trunk is rigid and the legs are thrown out in any direction. The cerebellar gait is often described as a drunken, reeling or titubating gait. It is, however, not like that of a drunken man who reels in any direction, but does this largely because of his cerebral incapacity. A slightly intoxicated person reels forward or to one side, while one who is much intoxicated is unable to stand and any attempt to do so brings about a collapse of the body. There is no attempt on the part of the drunken man to hold his arm outstretched for protection, whereas the cerebellar patient is always careful as to the manner in which he walks, for knowing his own tendency to stagger, he makes efforts to obviate this by assuming corrective positions. In other words, he calls upon his cerebrum for compensations which are largely out of commission in the intoxicated person.

Asynergia in Movements not Concerned in Equilibrium—Dysmetria: Asynergia also appears in movements of the limbs not directly concerned in the maintenance of equilibrium. In performing the finger-to-nose test, the act may be finally executed

by the patient, but the circle described in the entire movement is greater than normal and the component movements are quicker than usual. This defect is due to improper measuring of the movements, or dysmetria. The more marked the dysmetria, the greater the tendency for the sweep of the arm to be inward, due to the involvement of the shoulder girdle. Evidence of dysmetria in the legs obtained by the heel-to-knee tests is seen first in overflexion of the thigh on the abdomen so that the heel overshoots the desired mark.

Asynergic Speech Defects: Asynergia may likewise affect speech, giving rise to involvement of the tongue, face and laryngeal movements. Speech becomes jerky and irregular. The jaw muscles, the muscles of the pharynx, and the muscles producing movements of the head may be similarly affected.

Oculomotor Asynergia: The muscles producing movements of the eyeballs are frequently involved. As a result of such disturbance, the eyes are never at rest; they move upward or downward or to either side, it may be combining all of these movements with a nodding of the head. No two successive movements of the eyeballs are quite alike in cerebellar nystagmus.

Cerebellar Localization on the Basis of Clinico-pathological Evidence: Mills and Weisenburg arrived at the conclusion that there is a definite functional localization in the cerebellum. (Figs. 6 and 7.)

The culmen and the anterior lunate lobules are centers for synergic control of the head and eyes, the face and jaws, palate, larynx and pharynx.

The clivus and the entire inferior vermis of the cerebellum controls trunkal movements.

The posterior lunate lobule is the center for the upper extremity, its most lateral portion having control of abduction, its more mesial portion of adduction, and its intervening areas of extension of the limb.

The inferior portion of the cerebellar hemisphere controls the lower extremity,

the lobulus postero-inferior having control of abduction, the lobulus biventralis controlling adduction.

The rôle of the cerebellum is best exemplified in the performance of such complex

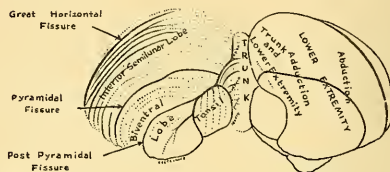


FIG. 6. SCHEME OF LOCALIZATION IN THE HUMAN CEREBELLUM; ZONES AND CENTERS OF THE SUPERIOR SURFACE OF THE CEREBELLUM, ACCORDING TO MILLS AND WEISENBURG.

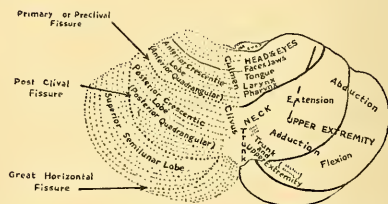


FIG. 7. SCHEME OF LOCALIZATION IN THE HUMAN CEREBELLUM; ZONES AND CENTERS OF THE INFERIOR SURFACE OF THE CEREBELLUM, ACCORDING TO MILLS AND WEISENBURG.

movements as those of fencing, boxing or in playing tennis. In the human cerebellum there are centers for simple synergic movements as well as zones for more complex synergic performances. It is important to recognize that each simple synergic movement has its special cortical representation in the cerebellum.

Synergia Comprehensive of Cerebellar Functions: The accumulated evidence obtained from the extensive investigation of the cerebellar problem seems at length to be harmonized in the conception that synergia is the principal function of the cerebellum. This determines a common

ground upon which all may stand, since the term *synergia* furnishes a definition of the functional attributes of the cerebellum in which the many differences in viewpoint may finally be conciliated.

Analysis of Synergia into its Component

Factors: Although it is not difficult to appreciate the general significance of synergia, its more intimate nature requires the further elucidation to be obtained from the analysis of its exact anatomical and physiological factors. Recent work by Adrian Lambert and Tilney¹⁸ has been carried on to this end. The material studied consisted of normal individuals and patients suffering from cerebellar disease. The method of investigation was a combination of *kymographic analysis with bradykinetic analysis* in which slow motion pictures were employed. The photographic rate of these motion pictures was 160 to the second instead of the usual cinematographic rate of 16 to the second. This method offered an opportunity to study normal and disturbed motion by means of an actual graphic record with a visual analysis of them by the slow motion pictures. As a result of these investigations, it was concluded that *synergia is dependent upon two factors:*

First, the establishment and maintenance of synergic muscular units throughout the body, and,

Second, the establishment and maintenance of coordination between the synergic units in the performance of complex acts.

The Synergic Unit: The conception of the synergic muscular unit was obtained by kymographic studies which showed that throughout the entire body the muscles are arranged in unit groups, each unit consisting of agonist and antagonist. The teaching, however, that these groups operate as antagonists has been misleading. They are antagonists only in the sense of their anatomical positions. Physiologically they are synergists and carry on their work by a mutually coordinated coopera-

tion. If, for example, a single antagonistic group be considered, such as the flexors and extensors of the wrist upon the forearm, it is apparent from their position on the dorsal and ventral surfaces that these muscles are antagonistic. When, however, their action is recorded by means of kymographic tracings, showing the movement of the flexors and extensors simultaneously, it at once becomes evident that they no longer may be regarded as antagonists but are in fact synergists. In flexion of the wrist, although the flexor curve rises to a higher point, it is followed throughout its entire course by a corresponding extensor curve, and similarly, during extension, although the extensor curve is the greater, it is none the less certainly followed by a corresponding flexor curve during its entire course. It thus appears that the flexors and extensors of the wrist are in reality a synergic unit and that when one acts the other acts with it.

The Dominant and Check Elements in the Synergic Unit: In this unit two factors may be recognized, first, the dominant element which determines the direction of the movement, and second, the check element which works constantly with the dominant in order to provide it with the necessary limitation so that the resultant movement may at no time be excessive but always exactly adapted for the purpose in hand. During flexion or extension of the wrist there is a constant relation between dominant and check elements. When the proper ratio between these two elements is lost, the synergic unit is then deprived of its even mode of operation and the movements in the parts become irregular. Extension becomes excessive because the flexor check element is not sufficient to meet it. Over-correction on the part of the flexors is therefore required, which in turn has an inadequate extensor check. This dissociation of the dominant and check elements leads to a series of irregular oscillations, constituting the clinical symptom

of ataxia which is based upon a physiological disturbance of the muscles. What is true of this simple group of muscles in the forearm is true of all the muscle groups in the body.

Simple Synergia: Every agonist has its antagonist and the two together constitute a synergic unit. There are synergic units in the hand, in the forearm, in the arm, in the shoulder girdle, in the trunk and neck, in the tongue and larynx, in the masticatory musculature and in the lower extremities. Each of these synergic units must be maintained in its proper relation in order that the volitional acts calling upon these groups of muscles may be carried on in their proper manner. When the proper relation between them exists, the muscles are in a condition of eusynergia, but when this relation is disturbed, defects in motion appear which represent a state of asynergia.

Integrative Synergia: The establishment and maintenance of coordination between the various synergic units which enter into the performance of complex acts is also an important feature in the mechanism of synergic control. The simple act of flexion and extension of the wrist requires but a single synergic unit. Other acts in themselves not extremely complex require the combination of several synergic units, such, for example, as lifting the hand to take a glass of water from the table, raising it to the lips, drinking and putting the glass again upon the table. This motor performance requires a coordinated operation of synergic units in the hand, forearm, arm and shoulder girdle. It necessitates the proper correlation of these units one with the other. They must act together and maintain the proper relations in the phases, rate, force and extent of the muscular contractions involved.

Kinetic and Akinetic Phases of the Synergic Units: During such an act each of the synergic units involved in the performance passes through two phases. At

one instance, it will contract to produce motion in the part, such as the contraction of the extensors of the fingers, opening the hand preparatory to grasping the glass or the closure of the fingers about the glass. Immediately these acts have been accomplished, the fingers remain flexed and motionless, although the muscles of the fingers are in active contraction. This illustrates the two phases through which all synergic units are constantly passing during volitional activity. The first, that which produces motion, is the kinetic phase; the second, that which produces a fixed position under active contraction of the muscles, is the akinetic phase. These two phases of each synergic unit must be correlated in the time of their action so that, for example, the fingers shall not close prematurely before the glass is grasped and also that they may pass at the proper time into the akinetic phase in order to retain the glass in the hand. The synergic unit must be correlated in the force applied during both the kinetic and akinetic phases. This must be adequate to the purpose of the act. The extent of the muscular contraction must likewise be regulated so that it fulfils the purpose of the act, otherwise the distances through which the part moves will be incorrect, with the resulting symptom of dysmetria, that is, overmeasuring or wrong measuring of the distances concerned in the muscular performance.

If in this relatively simple volitional act, it is necessary to coordinate the number of synergetic units mentioned, how much more complex is the synergy which brings into action the greater number of volitional performances, such as in running or leaping, in playing tennis or baseball, where all of the synergic units in the body come into play and must be maintained in their proper relations. The maintenance of the synergic units in proper coordination during the performance of complex acts may be called composite or "integrative synergia."

It is evident, however, that the basis of

all of these synergized movements is the synergic unit and that disturbances in it will lead to marked disorders of motion either in the entire body or limited to certain parts of it.

Types of Integrative Asynergia: Several types of integrative asynergia may be recognized, depending upon the distribution of the disturbance. Asynergia may be confined to the extremities, either to the arms or legs or to one extremity. This is *appendicular asynergia*. On the other hand, the defects in the synergic units may involve the axial musculature and the muscles of the trunk. This is *axial and trunkal asynergia*. A third type of asynergia affects both the extremities and the trunk simultaneously. This is *axio-appendicular asynergia*.

Synergia in the Light of Cerebellar Histology: An interesting light is shed upon this conception of synergia by the histology of the cerebellum. It is possible that provision is made in the histological connections of the Purkinje cells for the varieties of synergic control which have been recognized in the types of asynergia just described. It seems fair to presume that the "climbing fibers" which confine themselves to the trunks of the dendrites of single Purkinje cells have to do with the regulation of the simple synergic units. The "basket fibers" make possible the grouping together of collections of Purkinje cells whose purpose it may be to mediate the more complicated synergic control for the operation of groups of synergic units, while the connection accomplished by means of the "granule cell" brings about an assemblage of vast numbers of Purkinje cells in control of large numbers of synergic units, perhaps corresponding to the entire musculature of the body. In this sense, while it is undoubtedly the case that there is definite localization within the cerebellar cortex, this localization is by no means as discrete as that seen in the cerebral cortex. In fact,

it may be regarded that the cerebral cortex in its relation to voluntary motion has the supervision of motor purpose as its chief object, while the synergic control of the cerebellum has as its primary function the regulation of the muscular patterns necessary for performance of that purpose.

It is not surprising, therefore, that in the efforts to determine an exact functional localization in the cerebellar cortex so much more difficulty has been experienced than has been the case in the cerebral cortex. Cerebellar localization implies much more the patterns of movements than the parts employed. It is exact for circumscribed or limited acts, but it is diffuse and probably without definite limits for the great majority of volitional performances carried on by the muscles.

Summary Concerning the Functions of the Cerebellum:

1. The fundamental cerebellar function is synergia, which depends upon the proper relations of the synergic units of the body.
2. A synergic unit consists of a muscular group of anatomical antagonists whose physiological action is represented by the mutual synergism in a dominant and check element. The entire musculature of the body is divided into synergic units.
3. Simple synergia depends upon the proper correlation of simple synergic units.
4. Integrative synergia depends on the proper correlation of many groups of synergic units.
5. Disturbances of simple or integrative synergia lead to the clinical phenomenon of *asynergia or incoordination which is the essential cerebellar symptom*.
6. Asthenia and atonia are symptoms secondary upon asynergia.
7. The synergic control of the cerebellum is essentially ipsilateral, that is, for muscles of the same side of the body, but axial and trunkal muscles are under a bilaterally dependent control, both halves of the cerebellum being active in the regulation of their movements.
8. The synergic control of the appen-

dicular muscles is both bilaterally dependent and unilaterally independent, the unilateral independence being more prominent in the arms than in the legs.

9. The localization of definite areas in the cerebellum for the control of the several parts of the body is much more diffuse than is the case in the cerebral cortex, since the cerebellum regulates the entire motor pattern of the act, while the motor cortex of the cerebrum controls its purpose.

10. The superior and inferior vermis control the synergic action of the axial and trunkal muscles.

11. The superior vermis controls the movements of the eyes, neck, jaws, face, tongue, larynx and pharynx.

12. The inferior vermis controls the

muscles of the trunk and also acts in control of the bilaterally dependent movements of the extremities, that is, those movements of the limbs in which the extremities of one side act synchronously with those of the other side, as in walking.

13. The cerebellar hemispheres control the synergic action of the extremities in bilaterally independent acts, as, for example, in the movements of one arm alone.

14. There may be areas which regulate adduction, abduction, flexion and extension as such, but these are to be regarded as part of more comprehensive regions controlling movements of the extremities in all planes produced by the action of the synergic units.

PART III.

THE PRINCIPAL CEREBELLAR SYNDROME AND ITS VARIATIONS

PRESENTED IN COLLABORATION WITH DR. MARY JARVIS

The clinical records of diseases affecting the cerebellum comprise an extensive assortment of symptoms attributed to defects in this organ. Many of these symptoms are, in fact, extracerebellar, and not in the strict sense part of the *cerebellar syndrome*. Cases in which the lesion is limited to the cerebellum itself, although relatively rare, afford the best clinical opportunity of investigating the nature of disturbed functions due to intrinsic cerebellar disorders. The following case offers a suitable clinical example of this kind:

I. HISTORY.—The patient, a man forty-one years of age, unmarried and without occupation, complained of a severe motor disturbance which rendered him incapable of securing employment or of earning his living. He was dependent upon an unmarried sister who for many years had endeavored to find some means of relief for her brother.

Mentally, the patient was alert and normally active. His education had been neglected largely because it was impossible for him to attend public school on account of his motor disorder and also because the family did not possess the means to provide private instruction. For what learning he had acquired he was indebted to the patient assistance of his sister, who had taught him until he could read in a somewhat slow and hesitating fashion. This was the extent of his book learning. In the common knowledge of his walk of life he was not below the average, although he had never been able to associate with other men or participate in their usual activities.

His motor disabilities had occasioned many falls and bruises as well as numerous unpleasant experiences. It not infrequently occurred that his reeling gait and general unsteady appearance led to his arrest on the charge of intoxication, and

the fact that his intoxicated state was persistent, showing none of the usual signs of improvement at the proper time, puzzled the officials. While in charge of the authorities, it was admittedly impossible for him to obtain intoxicants, yet the signs of intoxication continued unchanged. The case always proved too much for police intelligence and the patient was finally discharged with the advice to see a doctor; but even this proved unavailing.

Often he applied for employment, but because of his unsteadiness of speech and manner he was usually rejected. Several times, however, he managed to obtain a position as one of a large group of laborers, but always with the same result. He could not do the work, or else in his vain attempts he would fall and injure himself.

His disease first manifested itself in infancy. He was unusually late in sitting up and standing. He did not learn to walk until he was five years old. His earliest attempts to move his hands and arms, to sit up or to rise to a standing position, were attended by irregular swinging movements of the arms, legs, head and trunk. In his early attempt to walk he could not take more than two or three steps without falling and never learned to run without losing his balance.

His speech at first was slurring and unintelligible, except to his sister. Ultimately it became clearer, but was jerky and irregular. He was unable to use his hands in any act which required steadiness and precision, because on such attempts his arms would begin to oscillate in such a way that he could not control them.

His general health was good. He complained of no pain or other distress. So far as he knew, with the exception of his muscular unsteadiness, his functions were all normal.

The family history of the patient con-

tained no record of a similar condition in any of the direct or collateral branches of his family. His sister, his only living relative, was a normally developed and capable woman who gave no evidence of any disturbance which in any way resembled that from which the patient suffered.

II. EXAMINATION.—At the time of the examination made in his forty-first year, he appeared upon inspection to be a man fairly well nourished and evidently a little older than his given age. He was six feet one and one-half inches tall; his weight was good. He answered questions intelligently, was mentally alert and co-operative. The neurological examination made on this occasion showed the following facts:

The Somatic Motor Component: This component showed that the *idiodynamic control*, as well as the *tonic control* of the muscles, was normal. The reflexes showed but a single departure from the normal. Upon taking the knee-jerk, with the patient sitting on the edge of the table in such a way that both legs hung free, the percussion on the patellar tendon caused an active response in which the leg oscillated seven or eight times backward and forward before coming to rest. All of the other deep reflexes were normal in their activity and equal on the two sides. There were no pathological reflexes and the superficial reflexes, including the upper and lower lateral abdominals and the cremasterics on both sides, were present and active. There was no demonstrable change in the tone of the muscles in any part of the body.

The volitional control of the muscles was normal on both sides and there was no appreciable decrease in the muscle strength of any muscle group. Paralysis and asthenia as a result of the tests made were found to be absent.

Skilled performances were much impaired. This, however, was not explainable on the basis of any volitional motor disturbance but rather in the distribution of the motor impulses by means of which the

synergic control of the muscles was maintained.

The Gait: In testing locomotion, the four tests of Fournier were employed. The first, which requires the patient to rise from a chair and begin to walk upon command, causes the sudden introduction of the equilibratory mechanism in locomotion. The patient upon rising from the sitting posture at once began to stagger; as he continued to walk, the staggering became more pronounced. His gait soon became reeling like that of a drunken man, now to one side, now to the other.

Upon applying the second test of Fournier, that is, having the patient stop quickly on command while walking, he showed a typical disturbance in his equilibrium. In attempting to stop he lost his balance and fell.

The third test of Fournier requiring the patient while walking to turn either to the right or to the left also brought out marked disturbances of equilibrium.

The staggering gait corresponded to the locomotor disturbance described by Duchenne¹¹ as *titubation*; but on further analysis it appeared that when walking, his legs at times seemed to precede his body which bent over backward or to one side, while at other times his trunk became flexed forward, in which case he would hasten his steps in order to keep his legs in line with the rest of his body. This was due to a decomposition of the synergized movements necessary to walking. Such decomposition of movement in walking is described by Babinski as *Asynergia Major*.

On attempting to run the patient invariably fell forward and to one side. In falling his hands were automatically thrust out in front of the body to break the force of the fall. Tested on going upstairs, he presented the same tendency toward loss of balance.

In attempting to rise after a fall he assumed a position upon hands and knees, and while trying to get upon his hands and feet his body would sway from one side to the other, often resulting in a complete

loss of balance. The lack of synergic control in his trunk muscles was pronounced, and his trunkal asynergia in all types of locomotion, whether upon his feet or when attempting to creep upon hands and knees, was one of his most prominent symptoms.

Station: In testing his station, he showed difficulty in standing with his feet close together whether his eyes were open or closed. In many cases of ataxia due to other than cerebellar conditions, it is a rule that the patient can manage to stand better with his eyes open than when closed. This is due to the fact that the visual impulses augment spatial orientation to such a degree that they make partial amends for these defects in muscle, joint, tendon and bone sensibility. In cerebellar lesions, however, it makes slight difference whether the eyes are open or closed, since the visual impulses compensate but little. In standing the patient always assumed a position with his feet far apart, but even under these conditions his body swayed from side to side or backward and forward in an irregular manner.

On examining his synergic control in acts not directly associated with equilibrium, such as the finger-to-nose test, the hand and finger on approaching the nose presented several irregular oscillations which gradually increased toward the end of the act. The finger was finally placed either in the eye, upon the cheek or on the chin. The oscillations in the upper extremity during this test were due to a lack of proper balance between the synergic units of the arm. It was impossible for the patient to perform this act correctly, regardless of the number of times it was repeated. Tests to demonstrate the synergic control of the lower extremities developed similar results.

Pass-Pointing: The attempt to touch a fixed point with the tip of the finger in any position in front of the patient consistently resulted in failure. This was the *spontaneous deviation of Barany*, which was not affected in any way by the production of artificial nystagmus. All of these defects in

synergic control were attributed to dissociation of the synergic units in the arms and legs which are necessary to the performance of the given acts.

The patient also showed marked defects in the integrative synergy of all movements of the trunk and extremities. He was examined by five tests devised by Babinski to demonstrate what he terms *Aasynergia Minor*. These tests gave the following results:

1. *Leg-Elevation Test:* The patient, in a supine position, was instructed to elevate the right leg. In doing so he began first by flexing the thigh at the hip and finally extending the leg from the thigh, resolving the act into its two component movements instead of performing it in one act, as is normal. This test was repeated with similar results in the left leg.

2. *Sitting-up Test:* The patient, in a supine position, was instructed to sit up. In complying, flexion of the body began before flexion in the legs. Normally these two acts are carried on simultaneously, but in this patient there was a dissociation of the components of the act resulting in actual decomposition of the movement.

3. *Foot Test:* The patient, while seated in a chair, was instructed to touch the hand of the examiner with his toes. This resulted in a series of irregular oscillations during which flexion of the thigh began before extension of the leg and foot, thus resolving the movement into its component parts. Both legs gave the same results.

4. *Kneeling Test:* The patient, standing beside a chair with his hands grasping the back of it, was instructed to place his knee on the chair. This he did first by flexing the thigh on the hip and then beginning flexion of the leg upon the thigh, thus dissociating the two movements. Both legs showed the same defects.

5. *Leaning Test:* The patient, in a standing position, was instructed to lean backward as far as possible. He was assured that he would not be permitted to fall. In leaning backward, extension of the spinal column was carried to a fair degree,

but the patient soon lost balance and would have fallen had he not been supported. There were no associated movements in the legs in this act. Normally, when a patient leans backward as far as possible, in order to compensate for the disturbance in the center of gravity in the body, the legs are flexed at the hip and knee. The two acts of extending the spine and flexing the lower extremities are essential to the maintenance of balance, but in this patient there was an evident decomposition of the two movements.

These tests furnish clinical evidence that the acts entering into any voluntary performance may be decomposed into their several component movements, thus giving rise to a lack of integrative synergia which is one of the most prominent of cerebellar symptoms.

Dysmetria: In the performance of all skilled acts, it was found that the patient manifested a defective judgment in the distance, force and time of muscular contraction to such a degree that the accomplishment of the purpose was often defeated. His movements were executed without the proper measurement in time, distance, rate and force in action. The initial impulse was either too strong, the movement too rapid or else tardily executed. This motor disturbance has been called *dysmetria* by André-Thomas. In three tests for dysmetria the patient showed the defect to a marked degree:

1. *Test of Grasping:* The patient was directed to lift his hand and grasp a glass of water on the table. The movement upward carried his hand well above the table. The fingers of the hand were opened before they needed to be. The hand came into contact with the glass so forcibly as to knock it over. The patient made several unsuccessful attempts to grasp the glass. This test showed that he had no proper measurement in the muscular activities necessary to grasp the glass.

2. *Test of Reversal of the Hands:* The patient was instructed to hold both arms extended in front of him so that the palms

of the hands were turned upward. He was then instructed to turn the palms down with as rapid a motion as he could. This resulted in a pronation carrying the palmar surfaces of the hands into a position of over-pronation.

3. *Heel-to-Knee Test:* The patient, in a recumbent position, was instructed to touch an object placed upon his right patella with the heel of his left foot. On the first attempt the left heel was carried well up on the thigh, while on the second it touched below the knee. The same disturbance was noted in the right leg.

Adiadochokinesis (a, privative; *diadocho*, succession; *kinesis*, movement): This is a symptom of cerebellar disease described by Babinski as a failure in the ability to produce succession movements. The patient was instructed rapidly to flex and extend the forearm upon the arm, but was unable to do so except in an awkward and irregular manner.

Rebound Phenomenon of Holmes: In testing the strength of the upper and lower extremities, the patient manifested a significant symptom. When the examiner grasped the wrist firmly and attempted to draw the arm into extension against the patient's resistance, suddenly releasing the wrist, the patient's hand rebounded so that it struck his cheek with considerable force. A similar rebound was also present in the lower extremity which would, if held flexed at the hip and knee by the patient against the examiner's attempts at extension, rebound forcibly when suddenly released and assume an extreme position of flexion. This rebound phenomenon of the extremities is another example of the dissociation between the synergic units, and demonstrated a failure in the check element.

Tremor: Every volitional movement of the patient was attended by a coarse, irregular tremor which was increased when he directed his attention to it or when he attempted to check it. The tremor was not apparent if the parts were not in action. It was also absent in sleep. It appeared upon the first attempt at voluntary move-

ment of the arms, from which, if the act were continued, it spread to the neck and trunk. It was noticeable in the head and trunk when the patient was standing or sitting. No other abnormal involuntary movements were present, and no abnormal associated movements were discovered by the examination.

The Cranial Nerves: The motor divisions of several of the cranial nerves showed marked defects of innervation.

Nystagmus: The entire oculomotor mechanism (Nn. III, IV and VI) was defective. The eyes were in constant motion, oscillating from side to side as well as upward and downward. The ocular movements, although irregular and jerky, were always conjugated. These movements were increased when the patient attempted to fix his gaze and became especially active when he looked to the right or left. This disturbance had existed since childhood without appreciable change. It gave the impression of a complete dissociation in the synergic units of the eyeball and is described as *irregular nystagmus*. The application of the calorific tests did not increase or in any way affect this nystagmus.

Disturbances of Cephalogyric Movement: Turning of the head either to the right or left and looking upward or downward, showed a marked tremor in the muscles of the neck, causing the head to oscillate in an irregular manner.

Asynergic Speech: The patient's speech was scanning, interrupted and sometimes explosive. Often his articulation was that of a stutterer. His words were syllablized or suddenly enunciated with unnecessary rapidity and force.

Asynergia in Mastication: When chewing the muscles of mastication showed a similar asynergic defect; the lower jaw was often drawn upward with unnecessary force or depressed to an unnecessary degree.

The first and second cranial nerves were normal. There were no defects of vision other than those occasioned by the constant nystagmus. The optic discs and

retinae were normal. There was no atrophy or pallor of either disc. There was no ocular palsy. The facial muscles were normal on both sides. There was no paralysis or disturbance of the soft palate, pharynx, larynx or tongue other than those described in connection with articulation.

Bradykinetic Analysis: The motor disturbances of this patient were studied by means of slow motion pictures which brought out more clearly the fact that all of his motor defects were due to an asynergia resulting from dissociation of the synergic units.

The Somatic Sensory Component: All qualities of special sensibility and general somesthetic sensibility were tested with care and found to be normal. There was no alteration of discriminative, tactile, cutaneous, muscle, joint, or vibratory sense. Pain and temperature sensibility was likewise intact.

The Splanchnic Motor Component: With the exception of the trapezius and sternocleidomastoid muscles, which showed asynergic defects already described, there were no disorders attributable to the splanchnic motor component.

The Splanchnic Sensory Component was normal.

Mental Status: Taking the patient's disadvantages into account, his mental condition was normal.

Laboratory Tests on the urine, blood and spinal fluid were negative.

III. INTERPRETATION AND ANATOMICAL ANALYSIS.—From the history and duration of the disease, it is clear that the lesion is in the nature of a congenital disorder.

Evidence of the focus of the lesion is furnished by symptoms, all of which are due to defects in synergic control and thus indicate an involvement of the cerebellum. A review of these symptoms demonstrates that they are essentially asynergic in nature. They are motor disturbances, all of which may be explained as failures in the proper action of the synergic units of the body. Analyzed from this point of

view, the symptoms align themselves as follows:

1. *The Pendular Knee Reflex*: This is due to an inadequate relation between the check and dominant elements in the synergic units which produce flexion and extension of the leg upon the thigh. Ordinarily the stimulation which causes the knee-jerk (percussion over the patellar tendon) stimulates the quadriceps extensor, the dominant element, and simultaneously the hamstring group which acts as the check element. Operating as a synergic unit, these two groups of muscles during the production of the knee-jerk, serve to limit the motor reaction to one or two oscillations of the leg, even when the initial excursion is large. This is due to the mutual check action of the two muscle groups concerned in the reflex, a fact which has been demonstrated by kymographic record in the case of all normal reflexes. When the synergic unit for extension and flexion of the leg is dissociated, the mutual check action is lost and the leg swings like a pendulum. The pendular knee-jerk in this case was not due to decrease of muscle tone, since the latter showed no appreciable change. All of the other deep and superficial reflexes were normal.

2. *Disturbances in Equilibratory Synergic Control*: The disturbance in gait shown by the oscillations when the patient was standing with the feet together or even when they were widely separated, was due to a dissociation of the synergic units necessary to the maintenance of the upright position. The disturbances of the gait in walking and running, in turning or going up and down stairs, were disturbances both in simple synergia and in the integrative synergia which resulted in decomposition of movement. The manner in which the body was inclined backward or forward in walking, thereby threatening to overthrow the proper maintenance of equilibrium, an example of major asynergia, affords another instance of decomposition in synergic movements. The disturbances in rising from a sitting posture and going

upon the hands and knees also show defects in the synergic units as well as in the proper combination of these units to maintain equilibrium.

3. *Disturbances of Non-Equilibratory Synergic Control*, as shown in the asynergia minor of Babinski, all illustrate a dissociation in integrative synergia. *Adiadochokinesis* is an illustration of a dissociation in synergic units, as is also *dysmetria* and the *rebound phenomenon of Holmes*.

4. *Tremor* of the intention type is due to dissociation of synergic units and in the strict sense, therefore, is asynergia rather than tremor.

5. *Nystagmus*, as well as the disturbances of cephalogyric movements, is the result of disturbances in the synergic units controlling the eye movements and the movements of the head.

6. *Disturbances of Articulation* indicate a similar disorder in the speech mechanism, showing that both simple and integrative synergia have become defective. The speech disturbance is, therefore, due to asynergia.

7. *The Disturbances of Mastication* illustrate the dissociation in the synergic units producing movements of the lower jaw.

By this analysis it is demonstrated that all of the motor symptoms were caused by defects in synergic control.

Evidence of circumscription of the lesion exempts all parts of the central nervous system except the cerebellum. The fact that *idiodynamic* and *tonic control* of the muscles is normal shows that the final common pathway is still effective. *Volitional control* is disturbed only in so far as the execution of volition is impaired by asynergia. The design and purpose of all motor acts is still intact and the strength of the muscular contraction is normal. The *automatic associative control* showed no disturbance. All forms of sensibility were normal. This is a fact of chief importance, since it establishes a differential distinction between the ataxia due to improper sensory perception from the muscles and that which

results from disease confined to the cerebellum.

IV. DIAGNOSIS AND PATHOLOGY.—This condition is due to a failure of development of the cerebellum which, in its pathology, shows an inadequacy in the chief cerebellar constituents.

V. NOMENCLATURE.—This symptom-complex is known as the *Syndrome of Cerebellar Agensis*. It is also termed the *Cerebellar Syndrome of Nonne*.

VI. VARIATIONS OF THE CEREBELLAR SYNDROME.—The cerebellar syndrome in the strict sense is observed only in those conditions which cause intrinsic defects limited to the cerebellar structure. It appears in its purest form in the agensis, as in the case just described. Other pathological processes, however, may produce the cerebellar syndrome. It may be caused by *Primary Progressive Cerebellar Degeneration of Holmes*. This is a familial disorder occurring in adults between the ages of thirty and forty, and progressing slowly to a fatal termination after a number of years. Clinically it is characterized by all of the symptoms of the cerebellar syndrome.

Atrophy of the cerebellum, pons and olives, known as the *Olivo-ponto-cerebellar Atrophy of Thomas*, may likewise show all of the symptoms of the cerebellar syndrome.

Certain familial and sporadic involvements of the spino-cerebellar tracts may present many clinical features in common with the cerebellar syndrome, but these symptoms are invariably combined with others which establish the differential diagnosis. Involvement of the spino-cerebellar tracts is attended with disturbances in somatic sensibility and frequently presents symptoms referable to disturbances of the upper motor neuron.

Extracerebellar Symptoms: Certain extracerebellar lesions which compromise the

cerebellum, or intracerebellar lesions which compromise the neighboring structures outside of the cerebellum, produce associated extracerebellar symptoms. Some of these extracerebellar symptoms are due to pressure upon the cranial nerves connected with the medulla oblongata and the pons Varolii. Among these are:

Failure of vision with choked disc due to increased intracranial pressure.

Vertigo and labyrinthine nystagmus due to involvement of the vestibular divisions of the eighth nerve.

Tinnitus and deafness due to the involvement of the cochlear division of the eighth nerve.

Facial paralysis due to involvement of the seventh nerve.

Internal strabismus due to involvement of the sixth nerve.

Dysesthesia, pain and paresthesia of the face due to involvement of the fifth nerve.

Paralysis of the muscles of the jaw due to involvement of the fifth nerve.

Anesthesia, pain and paralysis of the pharynx due to involvement of the ninth nerve.

Laryngeal paralysis, respiratory and cardiac disturbances due to involvement of the tenth nerve.

Spasm and paralysis of the tongue due to involvement of the twelfth nerve.

A good illustration of extracerebellar symptoms is afforded by tumors of the cerebello-pontile angle which, in addition to involvement of the seventh and eighth nerves, may extend forward to involve the roots of the fifth nerve or backward to involve the root fibers of the glossopharyngeus and vagus nerves.

Other extracerebellar symptoms are due either to pressure of masses outside of the cerebellum in the posterior fossa or else contained within the cerebellum itself and pressing upon the medulla or pons Varolii. These symptoms may include:

Upper motor neuron paralysis of the leg and arm on the side opposite the lesion due to compression of the pyramidal tract either in the medulla or in the pons.

Spasticity of the leg and arm due to pressure upon the pyramidal tract.

Convulsions, usually of the unilateral type due to irritation of the pyramidal tract.

Alterations in the deep reflexes either producing an increase or causing the appearance of pathological reflexes, such as the Babinski and ankle clonus.

Disturbances in somesthetic sensibility, involving the muscle, joint, tactile, pain and temperature senses.

Disturbances in associated ocular movements together with vasomotor, pupillary and secretory disturbances due to involvement of the sympathetic system.

Loss of muscular power.

Paralysis or asthenia is not observed in cases due to intrinsic lesions of the cerebellum, such as acute parenchymatous degeneration, chronic atrophy or cerebellar agensis.

Asthenia and atonia when present as a result of cerebellar disease are consequences of the involvement of an adjacent structure. These symptoms are most commonly observed in the more acute and fulminating affections of the cerebellum, such as tumor, abscess, gunshot wound and inflammation. It is questionable whether asthenia and atonia can be accounted primary symptoms in the cerebellar syndrome.

Cerebellar catalepsy, a condition in which the patient's limbs when placed in a certain position remain fixed for a considerable period of time, has been described as a cerebellar symptom by Babinski. This symptom, however, requires further observation before it may be attributed to defects of cerebellar function.

VII. SUMMARY.—The essential clinical features of the Cerebellar Syndrome are:

1. Asynergia as manifested in
 - a. The Pendular Knee-jerk
 - b. The Asynergia Major, which gives rise to the staggering gait caused by dissociation of synergic units of the trunk and extremities and the decomposition of synergic movements.
 - c. The Incoordination of Station, due to the same causes.
 - d. The Asynergia Minor, shown in the pass-pointing, finger-to-finger and finger-to-nose tests, and also in the five tests of Babinski for minor asynergia.
 - e. The Dysmetria, or improper measuring of extent, rate and force of volitional movements.
 - f. The Adiadochokinesis, a failure to produce succession movements.
 - g. The Rebound Phenomenon of Holmes.
 - h. The Tremor on voluntary movement which consists of irregular oscillations of the arms, legs and head.
 - i. The Irregular Persistent Nystagmus.
 - j. The Cephalogyric Asynergia.
 - k. The Asynergic Speech Disturbances, resulting in scanning, explosive and slurring articulation.
2. The absence of pronounced changes in the deep and superficial reflexes and in the tone of the muscles.
3. The absence of paralysis or actual loss of strength; i. e., Asthenia.
4. The absence of changes in general or special sensibility.
5. The absence of visceral disorders.

In brief, the symptoms of intrinsic cerebellar disease are confined exclusively to disturbances of the motor sphere and affect only the equilibratory and non-equilibratory synergic control of the muscles.

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ANEURISM IN THE POSTERIOR CRANIAL FOSSA

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Intracranial aneurysms, though not of uncommon occurrence, rarely give signs before rupture which allow of their diagnosis. As the diagnosis in the following case seems clear-cut, it is believed that a careful review of the history and neurological findings will be interesting.

HISTORY.—Patient: M. S. Nurse. Age fifty-three. Single.

Family History.—Mother died of childbirth at twenty-seven; father of pulmonary tuberculosis at forty-two. No brothers or sisters. Two half-brothers and one half-sister alive and well. No history of rheumatism, gout, Bright's disease, heart trouble, cancer, nervousness or insanity.

Personal History.—Born in Corning, N. Y. Has always lived in New York. Entered a nurses' training school at nineteen and has followed the profession of nursing ever since. Has always worked hard but has taken excellent care of herself. Habits of eating and sleeping somewhat irregular. Has taken no exercise except that in connection with her work. Takes one cup of coffee and about one cup of tea a day. No alcohol or drugs of any kind.

Weight.—Average weight 126 pounds. Best weight 138 pounds, twelve years ago. During past four months she has lost about 10 pounds. Present weight 117 pounds.

Previous Illnesses.—Until the onset of the present trouble she was always robust and well. She had measles, whooping-cough, chicken-pox and mumps in child-

hood. No other acute infectious diseases. On December 13, 1915, she had an operation for fibroid tumor, complete hysterectomy, ovariectomy and appendectomy.

Head.—Occasional headaches since childhood, occurring at intervals of about six months and lasting about a day, usually accompanied by vomiting. No headaches since the onset of the present trouble.

Eyes.—Vision excellent. For the past seven years she has worn glasses for close work. No diplopia or inflammatory disease.

Nose.—Not subject to colds. No nasal obstruction or epistaxis.

Ears.—No earache or discharge. Until the onset of the present trouble hearing was acute and there was no tinnitus.

Teeth.—Always kept in good condition. A number have been filled. No abscesses. Moderate pyorrhea checked by constant treatment.

Throat.—No sore throat, tonsillitis or hoarseness.

Respiratory.—No pain in chest, dyspnea, cough, hemoptysis or night sweats.

Cardiac.—No pain or distress over precordium; no palpitation, edema or cyanosis.

Gastro-intestinal.—Until the onset of the present trouble, appetite and digestion were excellent. No nausea or vomiting.

Slight tendency to constipation corrected by mild cathartic.

Genito-urinary.—No dysuria, nocturia or polyuria.

Venereal Diseases denied by name and

symptom. Two Wassermann reactions in 1915 were negative.

Menses.—Onset at thirteen. No dysmenorrhea. Periods regular until hysterectomy four years ago.

Neuro-muscular.—Until the onset of the present trouble, no nervousness, vertigo, tremors, convulsions or paralysis. Occasional insomnia when overtired.

Present Illness.—On June 26, 1915, as the patient was preparing for bed she felt a slight momentary tremor in the right side of her tongue. A day later a similar

and occasionally food regurgitated through the nose. About a week after the onset she began to lose her voice for periods of about four or five hours during which she could not speak above a whisper. This condition continued for nearly a year when she lost her voice entirely, and has never been able to speak louder than a whisper since.

About two months after onset she noticed a noise in the right ear which seemed like a "distant pumping machine." This was constantly present and gradually became louder until it sounded like "escaping steam." Later it became still louder and was likened to "terrific rhythmic pounding." About two months after the onset of the tinnitus she became absolutely deaf in the right ear.

In August, 1915, she was under observation at the Neurological Institute with a tentative diagnosis of syphilitic meningitis, but as lumbar puncture and Wassermann were entirely negative, she was discharged without a definite diagnosis. In November she went to Boston and consulted Dr. Cushing, who advised hysterectomy for removal of a fibroid, but gave no diagnosis as to the cause of her cranial nerve palsies. He expressed the opinion, however, that the condition was stationary and would not progress.

All symptoms remained stationary until June 30, 1918, when the entire right side of the face suddenly became paralyzed. On May 30, 1919, she suddenly developed a stiffness in the right side and back of her neck, and an area of exquisite, burning pain at the base of the skull to the right of the mid-line. From this time until the present there has been almost constant tonic-spasm of the muscles of the right side and back of the neck, which is so severe that she is unable to turn her head to the right. These spasms are accompanied by agonizing pain. For some time previous to the onset of this pain she had noticed an atrophy of some of the muscles in the right side of the neck. Ever since the onset of the pain she has had attacks of vomiting, nearly every day when the pain was most

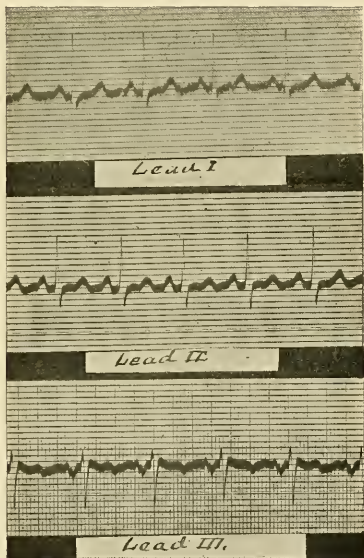


FIG. 1. ELECTROCARDIOGRAM SHOWING EXAGGERATION OF S DEFLECTION IN LEAD III, INDICATING PREPONDERANCE OF LEFT VENTRICLE.

spasm occurred, also two days later and four days later. The next day, three occurred. Seven days after the onset she noticed that she was unable to move her tongue as formerly and that it protruded to the right. Swallowing was difficult. She had attacks of coughing and choking

extreme. During the past two weeks the pain has been much less severe and there have been practically no vomiting attacks.

PHYSICAL EXAMINATION

The patient lies propped up in bed, turned partly on the right side, with her hands supporting the right side of the neck. There is no cyanosis, dyspnea or visible edema. Her general nutrition is fair. Mentally she is alert. The skin is soft and of normal color.

Head.—There are complete paralysis of the right side of the face and inability to close the right eye; obliteration of the

Nose.—There is no nasal discharge nor evident obstruction to breathing.

Mouth.—The lips are of normal color. The tongue is protruded to the right, there being a marked atrophy of the right half, which is also rather dry. There is no general or fibrillary tremor. Teeth are nearly all present and in fairly good condition. Very slight pyorrhea alveolaris.

Throat.—The uvula is in the mid-line. The tonsils are small. There is no pharyngeal injection or exudate.

Neck.—There is no thyroid enlargement, venous engorgement or abnormal pulsation.



FIG. 2. PHOTOGRAPHS SHOWING COMPLETE PARALYSIS OF RIGHT SIDE OF FACE AND RIGHT HALF OF TONGUE.

wrinkles on the forehead on the right side and of the right naso-labial fold. The mouth is drawn over to the left and the right cheek is relaxed and immobile.

Eyes.—Conjunctivæ and scleræ are clear. There is no injection or inflammation, no petechiæ, jaundice or arcus senilis. Pupils are equal and regular.

Ears.—Absolute deafness in the right ear. Hearing is normal in the left. There is no discharge from either ear or mastoid tenderness.

Superficial Lymph Glands not palpable.

Spine.—No spinal curvature, mobility normal. No vertebral tenderness.

Thorax.—Symmetrical; moves regularly and symmetrically with respiration; no abnormal pulsations. Breasts not tender. No masses.

Lungs.—Are normal throughout.

Heart.—Left limit of dullness is 9 cm. to the left of the mid-line in the fifth interspace. Heart sounds are of normal character. There are no murmurs. Pulse rate,

82, full, regular. Artery walls not palpable. Blood pressure, systolic 140; diastolic 90.

Abdomen.—Symmetrical, moves normally with respiration. Scar from old laparotomy in the mid-line. There is nowhere tenderness or muscular resistance. No masses or signs of peritoneal fluid.

Liver and spleen not felt.

Extremities.—No abnormalities of musculature, bones or joints. No edema or tremors.

9/15/19. *X-ray of skull* shows no intracranial abnormality.

9/22/19. *Report of Electrocardiogram.*—Curves representative of both auricular and ventricular complexes are normal in leads I and II. In lead III the deflection of S is exaggerated, which is indicative of preponderance of the left ventricle. The conduction (P-R) period is .18 second (normal).

NEUROLOGICAL EXAMINATION

September 4, 1919

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—Patient is in a reclining position, the head turned slightly to the right, the chin elevated. The head and neck are held rigidly in this position and the whole trunk is turned in looking to right or left. The right shoulder hangs lower than the left and there is atrophy of the muscles of the right side of the neck, and the upper portion of the right shoulder. The right side of the face is flaccid and immobile. There are no deformities of the spine or extremities.

Gait.—Patient walks easily without any evidence of disorder.

Coordination.—Equilibratory: Standing with feet together, eyes open or closed, there is no swaying. Non-equilibratory: The finger-to-nose, pointing and pass-pointing, and heel-to-knee tests are all well performed. There is no dysmetria or adiadochokinesis.

Skilled Acts.—There is no dyspraxia or apraxia. Patient is unable to speak above a whisper; otherwise there is no disorder.

Reflexes:

<i>Deep</i>	<i>Right</i>	<i>Left</i>
Jaw.....	2	2
Pectoral.....	2	2
Biceps.....	2	2
Triceps.....	2	2
Radial.....	1	1
Ulnar.....	1	1
Patellar.....	2	2
Achilles.....	2	2
<i>Superficial</i>		
Supra-umbilical.....	2	2
Suprapubic.....	2	2
Upper lateral abdominal.....	2	2
Lower lateral abdominal.....	2	2
Plantar flexion.....	Present	Present
Babinski.....	Absent	Absent

1 = slight; 2 = active; 3 = very active; 4 = with transient clonus; 5 = with permanent clonus.

Abnormal Involuntary Movements.—None noted.

Muscle Strength.—Neck: Patient is unable to rotate the head to the left more than about one-half the normal extent. Rotation to the right is normal. Elevation of the right shoulder is distinctly weaker than that of the left. Movements of the trunk and extremities are equal and normal on both sides.

Muscle Status.—Contour, consistence and tone are normal except for the right trapezius and sternomastoid.

Abnormal Associated Movements.—Not tested.

NERVE STATUS.—Not tested.

GENERAL SENSORY.—Touch, pain, temperature, vibratory, muscle tendon sense, are normal throughout.

CRANIAL NERVES.—*Olfactory Nerve and Nose:* Sense of smell seems normal in each nostril.

Optic and Oculomotor Apparatus.—Visual acuity in each eye, roughly tested, is normal. There is no contraction of the visual fields. Eye grounds: Discs are well outlined in each eye. Vessels appear normal. There are no hemorrhages or other abnormalities noted. Pupils are equal, regular, centrally placed, react actively to light and on accommodation. There is no exophthalmos, enophthalmos or ptosis. There is no strabismus or other ocular deviation. Nystagmus is absent.

Acoustic Nerve and Ear.—Patient is absolutely deaf in the right ear, no sound being perceived by either air or bone conduction. Hearing is normal in the left ear.

Trigeminal Nerve and Mouth.—Sensation of the face and mouth is normal. Movements of the jaw are normal and equal on the two sides.

Facial Nerve and Face.—Taste on the anterior two-thirds of the tongue on each side is diminished and uncertain to sugar and salt. There is complete paralysis of the right side of the face with absolute immobility of the forehead, orbicularis palpebrarum, and the muscles of the face including the platysma myoides. There is absence of secretion of tears in the right eye; also a diminished secretion of saliva in the right portion of the mouth so that it is drier than the left side.

Glossopharyngeus and Vagus Nerves, Pharynx and Larynx.—Taste on the posterior portion of the tongue is normal. Saliva secretion is diminished on the right side; probably normal on the left. The uvula is in the mid-line. Palatal and pharyngeal reflexes are active. Phonation: The patient is unable to speak above a whisper. Swallowing is said to be difficult. Respiration and cardiac action normal.

Spinal Accessory Nerve.—There is atrophy of the right sternomastoid and the superior fibres of the right trapezius so that the shoulder hangs somewhat lower on the right side. The outline of the back and upper portion of the shoulder are altered, being more concave than normal. The right shoulder does not move during respiration. The lower portion of the shoulder-blade is thrown outward and backward and is more prominent than on the left side. The middle portion of the trapezius seems practically normal.

Hypoglossus Nerve and Tongue.—The tongue is protruded markedly to the right, there being extreme atrophy of the right half of the tongue with wrinkling and deformity of the surface. There is no tremor. Articulation is good. Auscultation over the right mastoid process discloses a loud blowing murmur which is syn-

chronous with the heart beat. It is loudest at this point but is transmitted down the neck along the line of the carotid vessels for a distance of about 2 inches. Compression of the carotid artery against the carotid tubercle causes the murmur to disappear.

DISCUSSION

Aneurysms of the intracranial arteries are of quite frequent occurrence, although their presence is usually not suspected during life. Fearnside reports 144 instances in 5,432 autopsies. Osler found 12 in 800 postmortems; and Pitt records 23 in 9,000 examinations.

Etiology.—It is well known that aneurysm of the aorta and large elastic vessels are nearly universally of syphilitic origin. In a study of a large number of aneurysms Fearnside found that 96 per cent of all aneurysms of the aorta, 100 per cent of all aneurysms of the large elastic arteries, and only 6.25 per cent in the small muscular and elastic arteries were produced by syphilis. In the large elastic vessels the process starts by involvement of the vasa vasorum which leads to a disintegration and weakening of the elastic media and consequently a stretching of the wall and irregular enlargement of the lumen. Luetic involvement of the smaller vessels where the media is composed of both muscular and elastic tissue is quite different. Here the spirochete gives rise to an irritation and proliferation of the endothelium of the intima, the formation of granulation tissue and later fibrous replacement, which results in diminution in the vascular lumen and eventually an obliterating endarteritis rather than the aneurysmal formation.

Fearnside has given the whole subject of cerebral aneurysms very elaborate study and consideration and classifies the etiological elements somewhat as follows:

- I. Inflammatory: 1. Granulomatous (syphilis, tuberculosis, rheumatism, etc.). 2. Non-granulomatous (pyogenic infection).
- II. Non-Inflammatory: 1. Congenital. 2. Degenerative.

Aneurysms of inflammatory origin are usually of the non-granulomatous type. The infection of the vessel wall may take place either by direct infection from septic emboli within the lumen, by micro-organisms reaching the media or adventitia, by the vasa vasorum, by the settling of micro-organisms on the surface of the intima, or by direct inward spread of inflammation from surrounding tissue. Of these various possibilities, the infection due to septic emboli is probably the most common.

Aneurysms of intracranial arteries of syphilitic origin are uncommon. In the 44 cases studied by Fearnside there was no case of this group.

Vascular weakness resulting in aneurysmal dilatation is more frequently caused by non-inflammatory processes. The congenital group comprises those in which there is an inherent weakness of the elastic coat.

The non-inflammatory degenerative changes are frequently associated with high blood pressure and general cardiovascular disease.

The place of origin of these aneurysms is frequently at a point of branching or junction.

Distribution.—Von Hofmann studied 78 cases of aneurysm of the cerebral arteries and found the order of frequency of involvement of the various vessels as follows: (1) Middle cerebral; (2) internal carotid; (3) anterior communicating; (4) basilar; (5) vertebral; (6) anterior cerebral.

Symptoms.—In most instances there are no definite symptoms until rupture has occurred. While a rupture is usually fatal, with the general symptoms of cerebral hemorrhage, in a certain proportion of cases there is partial rupture or leakage at intervals with symptoms of repeated apoplectic attacks plus those of meningeal irritation. The removal of bloody spinal fluids on spinal puncture in a patient presenting these symptoms makes the diagnosis reasonably certain.

General symptoms of a brain tumor are rarely present before rupture has occurred. Many cases are reported, however, where there has been considerable leakage at

some time before death and in which headache and choked disc were present. Localizing symptoms before rupture are usually absent, but if they do occur, are due to irritation or paralysis of one or more of the cranial nerves. As the middle cerebral and internal carotid are the most frequent seats of aneurysms, the cranial nerves most often affected are the second and the third. Cases have been reported in which the fifth, sixth and seventh nerves have been involved, but I have seen no reported instances in which the tenth, eleventh and twelfth nerves were paralyzed.

Diagnosis.—The diagnosis of intracranial aneurysm in this case seems reasonably certain on consideration of the clinical history and neurological findings. The history is that of irritation and later paralysis of the right twelfth, eighth and eleventh nerves with paralysis of the seventh and involvement of the tenth on the same side. These lesions could conceivably be produced by a new growth, a basilar meningitis or an aneurysm of the vertebral arteries. It is not probable that a new growth in this locality, of four years' duration, could produce this group of findings without also causing compression of the medulla or cerebellum and signs of increased intracranial pressure. Basilar meningitis is certainly not likely in view of the negative serological findings. The presence of a murmur is not pathognomonic of an aneurysm, as it has been heard in other conditions. However, it is evidence in favor of an aneurysm, and taken in conjunction with the other findings, it seems quite probable that we have in this instance an aneurysm of the right vertebral artery originating at the junction of this vessel with its largest branch, the posterior inferior cerebellar.

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CHRONIC NON-DEGENERATIVE HEREDITARY CHOREA *

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The case about to be described presents several features which seem to distinguish it from the chronic, progressive, degenerative chorea, though the patient shows a clinical picture which closely resembles Huntington's disease. The question, therefore, is whether in the present instance we are dealing with a distinct clinical entity or an unusual form of Huntington's chorea. Not all chronic choreas, of course, are Huntingtons, and many cases have been recorded under various titles, such as chorea adulatorum permanens, chronic intermittent chorea, chronic perennial chorea, etc., but our patient apparently belongs to a special type of hereditary chorea, which is neither progressive nor degenerative. Before discussing the various features which tempt one to introduce a distinct clinical entity it may be well first to give a detailed description of the case, together with short histories of the ascendants, descendants, and collaterals in whom choreas have been found.

HISTORY.—R. P., American, female, married, age thirty-six, complained of peculiar movements of her arms, hands, body and legs, and twitchings of her face, all of which were aggravated by annoyance. She also complained of weak spells and pains about the heart. Her condition came on sixteen years before, was gradual in onset, soon assumed a stationary character and has not progressed for a number of years; in fact the patient believes that her condition is somewhat better. The onset is said to have followed upon a miscarriage, though the patient admits having had "shakings" of all parts of the body intermittently for a long time before that, but they had not interfered with her work and lasted for short periods at a time.

A little closer investigation shows that sixteen years ago, that is in 1903, the patient complained of weakness about the heart. While her husband was away from home she fainted and when found in that condition was taken to a hospital where she lay for two weeks, suffering from "nervousness" and weakness. She had many spells of weakness, but does not recall whether she had any irregular movements. She is quite sure, however, that the movements which she now presents were evident soon after she left the hospital. It is not certain whether the grimaces of the face, the movements of arms and legs and those of the body came on simultaneously, but they did not take long in reaching the present stage.

Some days the movements are better, at other times they are worse, but they never stop. They are present even at night during sleep. They are worse in the presence of strangers and when emotional demands are made upon the patient. The movements do not interfere with her work; occasionally she drops things, but only when very nervous.

Throughout her illness she has been suffering from spells of "weakness." These spells consist of having a dark cloud before her eyes, feeling faint and at times losing consciousness (?) and falling. She never has convulsions, sphincteric loss, biting of tongue, amnesia, etc. While "unconscious" she hears a buzzing sound, as if she were in the street. A quarrel, an injury or any excitement brings on an attack. There evidently is an important psychic or hysterical element, which may or may not have something to do with the patient's chorea; but its consideration will be deferred for discussion under diagnosis.

Past History is negative. Habits are

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good, sleep is disturbed by the "shakings," appetite good, bowels regular. Menses are regular, last six to seven days, are profuse and accompanied by cramps and headaches. She urinates six times by day and occasionally by night. Libido absent. Vision is poor, hearing normal, no vertigo, occasional pains in legs and back ever since the onset of her trouble. She claims that her memory is not good.

Family History.—Her father was the uncle of her mother. There is no history of epilepsy, insanity or alcoholism in the family. Her father had a similar condition to that of the patient. His arms and legs were shaking and he suffered from that condition for more than twenty years, or as long as the patient can remember. He is said to have dropped things because of his tremor, but its exact nature could not be determined. Mentally he was perfectly sound up to the day of his death, which was occasioned by tuberculosis at 68. One brother of the patient has shaking and trembling of both arms, but that does not interfere with his work, he does not drop things and mentally he is normal. Unfortunately I could not observe this brother although I have made several efforts to see him.

The patient married at sixteen, twenty years ago. The first daughter is eighteen, is not nervous but has poor eyesight and nystagmus. The second daughter is sixteen, has nervous spells during which she gets weak and her whole body shakes. The spells bear a functional stamp and the shaking could hardly be called choreic. Two miscarriages followed after the birth of the second child, but they were said to be due to "natural causes." Two boys, nine and six respectively, have chorea. Both have nystagmus. Their condition will be described later.

GENERAL STATUS.—The patient is very easily startled, the slightest noise or sound of voice, or movement intensifies her movements. Her gait is swaying, jerky, irregular, the whole body being incoordinate. There

is something clownish about it at times, and she often throws one side of the body forward as she makes a step. She cannot walk in a straight line, has difficulty in standing on either foot, cannot stand still for more than a moment and then only at the expense of great effort. Standing with feet together, eyes open and closed, brings out many irregular choreic movements of all parts of the body. Finger-to-nose, finger-to-finger and heel-to-knee tests are carried out with difficulty. There possibly is some dysmetria and adiadochokinesis. As for skilled acts, the patient has difficulty in picking up objects, owing to the chorea, but she never fails to get hold of them. Handwriting shows the effect of incoordination. The speech is slow, not scanning, but somewhat jerky.

Abnormal Involuntary Movements.—There are irregular, jerky, purposeless, meaningless, quick movements of whole parts, of arms, legs, body, hands. Occasionally twisting of the whole body. Cannot hold her head in one position for any length of time. The eyeballs wander in an irregular, jerky, choreic way in their sockets. There is something foolish and bizarre about all the movements. They are intensified by any emotion, they are temporarily absent when attention is diverted, activity brings them out, but a strong effort of the will momentarily controls them in one part, only to break out more violently in another. There is a coarse, irregular motor-facient tremor in the fingers, which may, however, be part of the chorea. In general, all the movements are more rapid than in ordinary chorea. The face shows an occasional grimace and the tongue is quickly jerked back on momentary protrusion.

The Superficial and Deep Reflexes. except for the abdominals, which could not be elicited on account of the flabbiness of the abdomen, are present and equal. No pathological reflexes. Muscle strength is normal, but she cannot maintain any voluntary effort for more than an instant. There are no atrophies or hypertrophies. The muscle tone varies from normal to hypo- or hyper-

tonus. Occasionally there were present uncontrolled associated movements during the execution of unrelated movements. All modes of sensation were normal over the entire body.

CRANIAL NERVES. *Vision.*—O.D. 20/100, O.S. 5/200, fields and fundi normal. Pupils were regular, equal, reacted promptly to light and on convergence. Eye muscles normal, but there was a general unsteadiness of movement of the eyes which was choreic in character and had none of the regularity or rhythm which is characteristic of nystagmus. Hearing was normal. Motor and sensory fifth (cornea) normal. The facial muscles were normal but showed choreic movements, occasionally forming grimaces. Palatal and pharyngeal reflexes present, uvula normal in position. The tongue was normal but could not be protruded for more than an instant, being immediately jerked back.

MENTAL STATUS perfectly normal. Despite the fact that the patient claimed poverty of memory she recalled things perfectly well and no real defect could be made out. What seemed to her poor memory is in reality defective attention. Emotionally she was easily irritable, quickly startled, and unduly annoyed, but she was not lachrymose or actually anxious.

SYSTEMICALLY, the examination proved negative.

Before discussing the case it may be well to describe briefly the status of two of the patient's children.

S. P., male, age nine and a half years, grade 3B, has twitchings and drops things. This began at the age of seven, when it was noticed that of a sudden he found difficulty in eating, buttoning his clothes, holding things, etc. Soon he could not feed himself, had difficulty in talking and walking and even wet and soiled himself. There were irregular, jerky movements of arms and legs, but few or no grimaces. The

condition was diagnosed as acute chorea; he was in the hospital for some time and altogether the severe movements lasted some six months. The condition never cleared up altogether, varied from time to time, but the boy was able to attend school. After two years an acute exacerbation occurred, which has lasted to date—about ten months—without any remission.

The past history is not significant. On examination the boy shows typical irregular, purposeless, jerky choreic movements of arms and legs. These are brought out markedly in all equilibratory and non-equilibratory tests. The deep and superficial reflexes are normal. There is irregular tonus of the muscles. Sensation is normal. The cranial nerves are normal. A quick, irregular nystagmus is present, possibly due to the fact that the boy is a partial albino. Systemic examination was negative.

A. P., the youngest boy, age six, has had slight irregular twitchings for three years. He has become restless, cannot sit for any length of time in one place or keep himself occupied. His past history is irrelevant, though he had measles, pertussis, diphtheria, pneumonia and pleurisy. Examination shows slight unsteadiness in equilibratory and non-equilibratory tests, but of a choreic nature. There are occasional mild purposeless, jerky, choreic movements in arms and legs. The reflexes are normal, muscle strength is good, sensation intact. All the cranials are normal, but there is some nystagmus on looking forward and trying to fix the gaze. General systemic examination is negative.

DISCUSSION AND DIAGNOSIS

To summarize, then, we have a young woman of thirty-six who has been suffering from a chronic, non-progressive chorea for sixteen years or more. Her father undoubtedly had a chronic chorea for twenty years, but apparently it was not degenerative in character. One brother has "shakings" of the arms. One daughter is

"nervous"; two boys have chorea, one of them a severe form which has lasted for almost three years with some mild remission, the other a continuous mild form, also for three years. The clinical picture of the woman is typically Huntingtonian in behavior and there is that bizarre clownishness about it which goes with that disease. There is no doubt about the hereditary nature of the condition, three generations showing chorea of a continuous and prolonged nature. But our patient does not show two of the most prominent symptoms which go with a Huntington. Her condition is not progressive, in fact is stationary if not somewhat better; secondly, there is absolutely no defective mentality, not to speak of even an approach to dementia. The poor memory which the patient complained of is certainly not due to mental degeneration, as her attention alone is defective.

Huntington's chorea is a hereditary chronic, progressive disease nearly always showing mental degeneration up to idiocy and dementia. It comes on between thirty and forty, at times after pregnancy. The clinical picture resembles chorea, except that the movements are somewhat quicker, the face shows more grimaces, the speech is rather differently affected, there is smacking of lips and tongue. The gait is clownish; there are rocking, flinging, suddenly-stopping movements in walking. The condition is intensified by emotion, but movements can be controlled by an effort of the will so that eating, writing, threading a needle and even shaving! may be possible. The inhibition of one set of muscles is usually carried out at the cost of restlessness of another set. The pathology of Huntington's chorea has not been quite definitely correlated with its symptomatology although various brain examinations revealed disseminated miliary encephalitic processes, chronic, diffuse meningo-encephalitis with atrophy of the cortex and changes somewhat similar to those found in general paresis. Some authors have found proliferation of neuroglia,

atrophy of supratentorial and radiating fibers and round cell infiltration about the vessels and pyramidal layers. Others think that the disease is due to congenital malformation of the motor cortex, with the above changes secondary in nature.

While the clinical picture of our patient fits in fairly well with that of a Huntington, serious consideration might be given to hysteria. The attacks of fainting or so-called spells of weakness speak in its favor, but there is nothing else either of psychic or somatic nature to confirm that diagnosis; nor would a hysterical condition be incompatible with chorea. It is a well-known fact that hysteria and chorea are sometimes associated. So too one need not lay too much emphasis on the marked intensification of the patient's movements by emotional stress, as this is in itself a part of most choreas and particularly of the hereditary type.

There are other forms of chronic chorea with which our patient's condition might be correlated, if it were not for a few outstanding contradictions which make it altogether unlikely. Chorea gravidarum is a remote possibility, as the condition began ostensibly with a miscarriage, but the fact is that that type of chorea usually stops with the emptying of the uterus and only rarely becomes chronic. Also it is never hereditary. Senile chorea is out of the question as it too is not hereditary and, besides, our patient is not senile. The father's condition might be classed with the senile choreas, but the hereditary nature subsequently revealed would render such a possibility unlikely. It may be added that senile chorea is frequently associated with mental symptoms, from which, however, the father was entirely free.

Paramyoclonus multiplex is a more likely possibility, as some of the movements of our patient might be so interpreted. However, there is not that lightning-like and universality of contraction in our patient which is found in myoclonia. The bizarre character of gait and facial and tongue movements and the hereditary

or familial attributes are lacking in paramyoclonus multiplex. The functional nature might well go with either condition, but cannot be relied upon as a deciding factor.

Chorea adultorum permanens, chronic perennial chorea, and chronic intermittent chorea are merely descriptive names of unexplained prolonged choreas, and are merely mentioned as unfit classifications for our patient's condition.

CONCLUSION

Critical analysis of the history of the case together with the clinical data force the conviction that we are dealing with a non-degenerative type of hereditary chorea. While there may be dispute as to the kind of chorea in the various members of the family scattered over three generations, there can be none as to the hereditary nature of the condition. It is, indeed, true that the children thus far present fairly common forms of chorea minor, which might be purely coincidental; it is hardly fair, however, to make such a statement

in view of the marked tendency to chorea in the family. Besides we are not at all sure that their choreas will clear up, and in view of the past history it would be rash to give a favorable prognosis.

Granting the hereditary nature of our patient's disease and seeing that the clinical picture is that of a Huntington, one might call it by that name in spite of the fact that it is not degenerative. But the utter absence of any mental change in the patient and the fact that the condition is not progressive would tend to differentiate it rather sharply from the common clinical type. Furthermore, while mental deterioration has very rarely been found wanting in Huntington's chorea it is not likely that that absence would characterize the choreas in all the members of the family and in all three generations. It is possible, of course, that our patient presents an atypical form of Huntington's chorea, but the greater likelihood is that we are dealing with a special type of non-degenerative hereditary chorea. Whether or not it is a distinct clinical entity is an opinion which does not as yet admit of proof.

TRAUMATIC NEUROSIS

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This study was undertaken with an open mind, and the cases were analyzed with a view to bringing out what actually could be observed rather than the establishment of some preconceived notion concerning the influence of trauma in the production of the so-called functional nervous disorders. The investigation was limited to the neuroses. No case was included which had any functional palsies, anesthesias, convulsive, somnambulistic or other hysterical manifestations. That sort of material will be left for investigation on another occasion.

The Material.—This consists of 102 cases which were personally observed by the writer. All of these cases were referred by the medical departments of various insurance companies or by the medical department of the State Industrial Commission of the State of New York. The patients were working men and women engaged in hazardous occupations within the meaning of the Compensation Law¹ of this State, and were not usually highly skilled technical mechanics or artisans. There is every reason to believe that the patients comprising the material investigated were honest and did not deliberately exaggerate their condition. In every case a complete neurological examination was made, blood pressure taken, fundi examined, and a complete physical examination was also recorded. A number of the patients were seen several times and many of them were treated for a period of months. Usually several months had elapsed from the date of the injury before the patient was first seen by me. No case was seen any earlier than three weeks after the injury.

The Observation.—Of the 102 cases investigated the average age was thirty-five and a half years; 84 were married and 18 single. The disproportion between married and single patients requires some explanation, and in view of the psychological considerations which will be discussed later the high incidence in married individuals is of some importance, especially in view of the fact that of the 102 cases only 10 were females. The suggested explanation for this is that married men confronted with the possibility of a long period of disability following an injury are much more apt to react with functional neuroses, particularly depressions, than would single individuals.

In all there were injuries to the head in 92 cases. In 72 of these the injuries to the head were in the nature of a direct trauma, that is, falling hammers or stones or bags of material, etc., which struck the patients on the head. In 20 cases the injury to the head was as a result of a fall of from 20 to 30 feet, the patient striking upon the head in falling. There were 28 of these falling accidents, but of these in eight cases the patients said that they struck on the back, indicating the lower dorsal and lumbar regions of the spine. There were two patients who were struck severe blows in the stomach in falling. The vast majority of the cases, therefore, consisted of patients who had experienced trauma to the head, usually sufficient to inflict at least one scalp wound. The injuries were all sustained in large factories where there was considerable noise and excitement following the accident, or the falls were from buildings where the mechanic was employed and where there was invariably excitement attending the fall. Of these patients, 54 were unconscious for periods extending from a moment or so to a half

¹The Compensation Law of this State (New York) allows a maximum weekly indemnity of \$15.00. Certainly this sum can be earned by the average laborer even though he is not particularly skilled. Besides this, many patients were married and had families and there was every reason for them to return to their work as soon as possible.

hour. Careful inquiries were made to exclude under this term those cases where a momentary stunning had occurred rather than real unconsciousness. It can be definitely accepted that in the 54 cases mentioned an actual loss of consciousness did take place. In 48 cases the injury was not sufficient to cause anything more than a momentary stunning. Without exception each one of these injuries excited in the patient at the time of the accident a really deep emotional reaction. They all confessed to profound degrees of fright. A great many of them volunteered the information at the time of their examination that they could not bear to think of going back to situations where a repetition of the accident was possible. A good many of them have consistently refused to return to the same sort of occupation that they pursued prior to the injury. It was observed as a regular thing that the patients who had sustained their injuries as a result of falling accidents gave the most marked neurotic reactions.

Some time ago, having been attracted by the statements of many writers, to the effect that the symptoms in the functional war neuroses nearly always followed a considerable period after the injury was sustained, I made very careful inquiry concerning what one might call the period of incubation. Particular stress, therefore, was laid upon gaining accurate information as to the length of time transpiring between the date of the injury and the date of the onset of the nervous symptoms. In 81 of my cases the symptoms began immediately after the accident. In 21 the onset occurred from two days to as much as 30 days (in one case) after the injury. The average was about 8 days. Therefore the so-called period of contemplation which was so common in the war neuroses and which is said almost invariably to have occurred following war injuries before the development of the neurosis, did not occur to any remarkable extent in these cases.

The number of complaints which the patients made were as follows: The most

common was sleeplessness, which was complained of in every case. The sleep was interrupted, not restless, and often punctuated by horrible dreams, which quite frequently had to do with the accident. These dreams were in the nature of a rehearsal of the traumatic experience and were attended by great fear. In 83 cases the patients complained of headaches which were poorly localized or not referred to any definite portion of the head. In 80 cases dizziness was also complained of. The dizziness was not a true vertigo and was in no case accompanied by vomiting, falling or staggering. Of the positive physical and neurological findings very little can be said. The examinations were consistently negative, except for the condition of the superficial and deep reflexes. These were without exception always active. This finding is not in my opinion to be lightly passed over. It is not common to examine 100 normal patients and find as a result active reflexes in each case. In normal individuals these reflexes can always be elicited, but in the patients under discussion not only were they elicitable but they were definitely hyperactive. Using the numeral 1 for normal (elicitable) reflexes, the reflexes in these patients may be expressed by the numerals 2 and 3. The reflexes, however, were invariably equal, and no case was found in which a Babinski or clonus or other pathological sign could be said to exist.

In passing, attention is called to the fact that these patients with but very few exceptions were hypersensitive to pain and temperature stimuli. Testing for the affective sensations gave responses to which an unpleasant element was added. There were no tremors, except a coarse, irregular unsteadiness in the extended hands so often seen in all types of functional nervous disease. There were no anesthetics and no motor symptoms of any kind.

Mentally no abnormalities were noted excepting that quite regularly there was a mild depression or anxiety state. It was

not often found that the patients complained of fatigue. They liked to be outdoors and many of them said that they felt better when they were out walking. The patients were usually restless and often volunteered the information that they felt like fleeing or screaming.

Attention to the cardiovascular system brought out nothing of note. The pulse was usually increased to 80 or 85. No abnormalities of heart action were noted. The average blood pressure reading was systolic 128 and diastolic 70. In two patients who did not otherwise show as a result of examination of either their fundal or peripheral blood vessels any evidence of arteriosclerosis or kidney involvement, the blood pressure was as high as 170 systolic, the diastolic readings in these two cases being 90 and 100 respectively. In one patient the blood pressure was 102 systolic and 66 diastolic and in another 105 systolic and 70 diastolic. The lowest blood pressure reading was 98 systolic and 58 diastolic. As a rule, however, the usual reading was about 130 systolic and about 80 diastolic.

Symptoms tending towards evidences of possible disturbance of the glands of internal secretion proved disappointingly absent. There was no case in which it could be said that there was a definite enlargement of the thyroid gland. None of my patients showed any of the eye symptoms of hyperthyroidism, nor did any of the cases show any other of the characteristic symptoms of this condition. None of them had either slight or considerable elevation of temperature. Only one patient had the so-called Sargent white line. No case presented itself in which sugar was found in the urine. There were none who had evidence of disturbance in the distribution of fat throughout the body, nor was there any evidence in any of them of abnormalities of growth or stature. There were no complaints of falling hair. A good many of the patients complained of subjective feelings of cold or heat in the extremities, and a very few showed objectively the

corresponding condition. In about 20 of the patients x-ray examinations had been made of the skull (sella turcica) and the results of such examinations were invariably negative.

The one fact that struck me in the observation of the facies of many of these individuals was their drawn, worried perplexed expression. Many of them corrugated their brows and faces and had the appearance that is so often noted in cases of melancholia. No photographs were taken of these patients, because it was impossible to obtain recent photographs of the individuals prior to the accident for purposes of contrast. Few of them thought they were ever going to be perfectly well, at least as well as they were before the accident, and many of them refused to believe that they were ever going to get well, and this mood seemed reflected in the faces of many of them.

Summary of the Type of Traumatic Neurosis.—The patient is usually a male, aged 35, married, who has suffered an injury either to the head or back. The accident itself is always attended with much emotional reaction, great excitement and fear. In a sufficiently large number of cases to make it noteworthy, there is a short period of unconsciousness. Usually the symptoms complained of begin very shortly after the accident, in about 20 per cent of the cases within eight days. The complaints made upon examination are usually a mild, depressed or anxiety state, sleeplessness, headaches and dizziness. Careful inquiry was made in each case as to the previous mental and physical condition of the patient. In no instance was it admitted that the patient had been nervous or ill, or had suffered from any mental disturbance prior to the accident. This statement will be startling, I am sure, to a great many. We have all of us grown to look upon these cases as examples of an abnormal reaction occurring in individuals predisposed by the so-called neurotic make-up or constitution which is said to be necessary for the development of func-

tional nervous conditions following injuries. Until these cases presented themselves, the writer also felt that this conception was correct. Reservations, however, will surely have to be made concerning this matter. I do not believe that many of the patients whom I examined tried to deceive me. They had very little or nothing to gain in the way of pecuniary profit. Certain it is that careful inquiries were insisted upon, and the fact that in no case was it possible to get a history of previous existing functional disorder is certainly quite striking. This one point, namely, the fact that in such a large group of cases a functional nervous disease can develop in individuals not previously mentally unusual, must necessarily presuppose a profound influence by the actual trauma or its attendant phenomena as a cause of the condition. In searching for an explanation of the symptomatology of these cases, investigation must lead to physiological, anatomical and psychological considerations.

The Anatomico-Physiological Explanation.—As the only possible anatomical investigation open to us in the functional nervous conditions following trauma are those which were conducted during the recent war in cases analogous to ours, reference is made to the work of Mott.² This study of Mott's deals with the effects of injuries in a group of cases in which there are "injuries of the central nervous system without visible injury." Among them are included the functional neuroses which alone concern us in this discussion. Concerning these Mott says that "they must be accompanied by chemical or physical changes in the nervous tissue, even though our methods of investigating the material do not enable us to say that the living matter was altered." In explanation of the symptoms seen in the functional neuroses, Mott adopts the theory of the "attraction and retraction of the dendrons." He explains the periods of unconsciousness on the theory that there is a

retraction of the branches of the intercalary neurones of the gray matter. He believes that these neurones have ameboid characteristics. He thinks that severe concussion causes "dissociation of the cortical perceptor neurones and produces a disturbance of consciousness." He refers also to the fact that a continuous supply of oxygen is necessary to the intercalary neurones in order to maintain consciousness. A violent emotion may, by its influence on the circulation and the vasomotor center, produce an immediate lowering of the oxygen content in the fluid and thereby suspend this function of the intercalary neurones. The symptoms of headache, anxiety, dizziness, fatigue, etc., he explains as due to a lowering of the neuropotential reserve, which is the result of insomnia, terrifying dreams, etc.

Mott is of the opinion that repeated and prolonged exposure to shell fire does produce a neurasthenic or hysteric condition in potentially sound soldiers. This must perforce be so in my cases of traumatic neurosis occurring in civil life, if the statements of my patients that they were previously perfectly well are reliable. Mott adds that the symptoms of shell shock, with the exception of profound unconsciousness in severe cases, accord with those of the common neuroses. It might be mentioned in passing that none of my cases had any of the degrees of amnesia so common in the neuroses following shell fire, a symptom which some of us met so often in the "shell shock" cases of the recent war.

Reference is made to the work of Pike and Coombs³ in their study of traumatic shock as having a possible bearing on the anatomical explanation of our cases. Two of the experimental facts published in their article follow:

"1. When the cells of the brain and medulla oblongata are deprived of blood for a period of from 10 to 20 minutes, a change in the staining reactions of the cells is demonstrable if the brain and upper

²The Effects of High Explosives upon the Central Nervous System. The Lettsonian Lectures. *Lancet*, 1916, Numbers VII, IX, XI.

³*Jour. A. M. A.* Vol. LXVIII, No. 25, June 23, 1917, page 1892.

portion of the spinal cord are removed some minutes after the circulation to the head has been restored.

"2. There is a greater susceptibility of these previously damaged cells to strychnin. Paralysis and failure of function from administration of strychnin during the resuscitation period occur more readily in the anterior (cephalic) portion of the central nervous system, which has been deprived of blood for a time, than in the posterior portion through which the circulation has been maintained continuously."

Perhaps reference should also be made to the work of Crile⁴ in which this author was able to show that structural changes occur in the cerebellum, suprarenal and liver of rabbits who were subjected to the influence of emotions of fear. These findings were in the nature of definite changes in staining properties of the brain cells and of the cells of the suprarenal and liver, besides which the normal arrangement of these cells was considerably interfered with. In discussing these matters Crile says that exertion, injury, the emotions, etc., produce histologic lesions identical with those found when adrenin is given for a long period of time. He states that extreme exertion, emotion and injury "cause histologic changes in the brain, the suprarenals and the liver, an increased output of adrenin, hyperplasia of the thyroid and a change in its iodine content, and changes in the glycogen content of the muscles and of the liver. In our patients no evidence of any dysfunction of the thyroid or of the adrenals was encountered. Whatever of Crile's theory of the Kinetic Drive is true, it does not apply in the cases under discussion with the possible exception of his findings as they refer to the emotions, particularly fear. In passing, I should like to call attention to the figure of a French soldier on page 2136 of Crile's article, which corresponds in great detail to the facies of several of my patients. In this connection, mention might be made

of sections from the brain of a French soldier who died of exhaustion following fatigue and prolonged emotional shock. The part sectioned by Crile was the cerebellum. The same changes in the staining qualities of the cortical cells were noted as were found in the cerebellum of the rabbit subjected to the influence of fright. Crile also found the same changes in this soldier's suprarenal and liver as he found in the animal. These matters are mentioned only in so far as they may have a bearing on the explanation of the symptomatology of the condition under discussion. Concerning the adrenals during emotional states, Cannon and De La Paz⁵ admitted that there was a discharge of epinephrin. Falta states that an increase in the adrenal secretion makes muscle hyperactive to stimuli. This is mentioned as a possible explanation of the increased activity of the deep reflexes present in my cases.

Nothing definite of course can be correlated between the disturbances of the thyroid and the adrenal and the condition under discussion. Surely nothing in the physical or neurological examination was brought out which would correspond to evidence of disturbance of these glands. It would appear that the theories of Crile and Cannon in this regard do not apply here, but it must be admitted, too, that these cases were seen a long time after the acute emotional shock. For observations of value on these points it would be necessary to see the cases soon after the accident, when not only physical and neurological examinations might be of value, but also chemical examinations of the blood. Cannon's recent discussion of the whole question of shock would seem to point to traumatic toxemia as the cause. It would not appear that any of the points scored by him in the elucidation of this problem can be made to apply as an explanation of the facts in our cases. It does not seem possible to draw any analogy between the conditions surrounding the onset of the symptoms in these patients

⁴Jour. A. M. A. Vol. LXV, No. 25, December 18, 1915, page 2130.

⁵Cannon & De La Paz, *Am. Jour. of Physiology*, 1911, Vol. 28.

with what happens when real surgical shock occurs. Hemorrhage does not occur sufficiently to cause any degree of shock, nor is there any infection to explain it. Cannon⁶ mentions the fact that some view shock from the standpoint that there is a general exhaustion of nervous cells. As he points out, however, shock can occur from injury to an extremity which has been cut off entirely from the spinal cord by means of section of the nerves. It would not appear, therefore, that the nervous system is essential for the production of shock. But he states: "It would be rash, however, to argue that nervous impulses play no part in the establishment of shock; indeed there are cases in which a pure nervous shock from trivial wounds seems to be present." He further says that these cases are very rare and lays stress on the fact that in the vast majority of cases shock is associated with severe tissue damage.

Psychological Considerations.—The matters treated above in the discussion of the anatomo-physiological points are of course quite indefinite and too much bound up in uncertainty to be helpful. It seems that our problem is essentially psychological. In considering the cases under discussion from this standpoint, one finds a ready explanation when the matter of a disorder founded upon instinctive reactions is considered. All of these patients at the time of their accident suffered the emotion of fear and were preyed upon by the instinct to flee from a hurtful situation. The instinct to flee and its attendant emotion, fear, are deeply rooted in all animals. Man, however, seldom experiences the emotion to a profound degree; but the instinct to flee is present and just as deeply rooted as it was in primitive times. When the instinct to flee was called up as a result of threatened injury from an enemy, man in his primitive state reacted promptly by setting in motion the mechanisms which accompanied the emotion fear and the

instinct was obeyed in terrified flight. As intellectual development following upon a change in environment gave man more perfect control of his instinctive reactions, flight became less desirable or possible, and so the emotion of fear and its attendant manifestations were inhibited through his experience. Physiological experiments seem to show without much doubt that certain of the glands of internal secretion are changed in their functions as a result of the emotions, and that the purpose of this change is to enable the body to mobilize energy, transforming it into the muscular movements necessary for flight or for fighting. When, however, flight is not possible or is inhibited, the emotion fear is experienced, and is accompanied by the physiological changes without the physical means of expending the energy mobilized, thus causing exhaustion without actual expenditure of muscular energy. This may be the explanation for the restless hyperactive condition of these patients. A further consideration of these instinctive reactions shows that the inlet for the instinct of flight may be by varied paths, as MacDougall says:

"Through injuries we may learn to fear or to be angered by the presence of a person or animal or thing towards which we were at first indifferent; and we may then experience the emotional excitement and the impulse to the appropriate movements of flight or aggression without recalling the nature and occasion of the injuries we have formerly suffered: i.e., although the idea of the former injury may be reproduced by the perception or by the idea of the person, animal or thing from which it was received, yet the reproduction of this idea is not an essential step in the process of reexcitement of the instinctive reaction in its affective and conative aspects; for the visual impression made by the person or thing leads directly to the excitement of the central and efferent parts of the innate disposition. In this way our emotional and conative tendencies become directly associated by experience with

⁶W. B. Cannon, *Jour. A. M. A.* Vol. LXXIII, No. 3, July 19

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⁷*Social Psychology*, Wm. MacDougall, John W. Luce & Co.; Boston, pages 38-55.

many objects to which we are natively indifferent; and not only do we not necessarily recall the experience through which the association was set up but in many such cases we cannot do so by any effort of recollection."

This summarizes the psychological explanation for the reaction exemplified in the cases above analyzed. In this way, too, one receives an acceptable explanation of why so many of these cases last for such prolonged periods of time. According to this explanation of MacDougall's many things may consciously or subconsciously reactivate by association the emotional terror surrounding the accident, and in that way continue to add to the mental condition which is responsible for the symptoms. One receives an insight into the reasons for the continuous depression present in these patients in this way that is not possible by the anatomical and physiological considerations alone. As MacDougall again says (page 42): "All the principal instincts of man are liable to similar modifications of their afferent and motor parts, while their central parts remain unchanged and determine the emotional tone of consciousness and the visceral changes characteristic of the excitement of the instinct." Therefore while the instinct to flee may arouse the emotion fear without expression through the bodily movements necessary for flight, nevertheless the emotional tone of consciousness is influenced, and the visceral changes which occur with the emotion continue and will remain indefinitely, activated each time the instinct is aroused, by conscious or by subconscious association processes.

The Outcome.—The average duration of the symptomatology in these cases was from six months to an indefinite period. A

number which have been followed through a period of three years did not clear up until the pending litigation before the State Industrial Commission was terminated. Interested in what took place after the termination of litigation, we conducted an investigation for the purpose of finding out when these patients resumed work after their cases were settled. It was thought best to choose a small number at first, so a group of twenty-five was investigated. From these twenty-three answers were received. One patient of the 23 still complained of many of the functional nervous symptoms and was not working. The other 22 had apparently recovered or at least were working and were not making further claims for compensation.

I do not believe these cases should be allowed to remain too long without serious attempts at resumption of their usual place in the environment. At the same time, until we learn a little more about their pathology and physiology, it seems best to recommend that sufficient time shall have elapsed for recovery from the initial emotional shock before insisting on this procedure. Exactly how long this period of rest and treatment should be, is impossible to say. Of late I have been recommending the return to work within a period of three months after the injury. It has been our experience that sooner or later in some of these cases factors connected with the possibility of financial gain as a result of the injury may enter into the psychological picture, usually through suggestion by interested relatives and friends. When this point has been reached it is often extremely difficult to readjust these individuals, and one occasionally finds that he has a patient so fixed in his neurosis that even a good settlement of his case is of no avail.

TRAUMATIC HYSTERIA

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At first, before a careful analysis of the material reported on in this connection and in that dealing with the cases of traumatic neurosis, it was intended to follow the usual procedure of grouping the cases. Early in the analysis of these cases, however, the writer became convinced that while it might not be absolutely certain we were dealing with separate entities, yet that the mechanisms precipitating the two types of reactions were different, and that the individuals manifesting them had inherent mental and physical qualities which differentiated them. The analysis of the 102 cases reported as suffering from traumatic neurosis¹ showed that these patients were usually males "aged 35, married, who has suffered an injury either to the head or back. The accident itself is always attended with much emotional reaction, great excitement and fear. In a sufficiently large number of cases [54] to make it noteworthy, there is a short period of unconsciousness. Usually the symptoms complained of begin very shortly after the accident, in about 20 per cent of the cases within eight days. The complaints made are usually a mild depressed or anxiety state, sleeplessness, headaches and dizziness. . . . In no instance was it admitted that the patient had been nervous or ill or had suffered from any mental disturbance prior to the accident. . . . This one point, namely the fact that in such a large group of cases a functional nervous disease can develop in individuals not previously mentally unusual, must necessarily presuppose a profound influence by the actual trauma or its attendant phenomena as a cause of the condition." It seems to us that this summary of the cases which we have included under the classification "Traumatic Neurosis," points to a definite group.

The most striking fact about these patients is that none of them at any time showed any of the usual gross physical signs of hysteria. I refer to palsies, anesthesias, aphonia, astasia-abasia, amaurosis, deafness and convulsive seizures. Added to these purely physical characteristics of hysteria, one must include also the symptoms of hallucinosis and somnambulism and also amnesic states. In this group of cases reported as "Traumatic Hysteria" one of these symptoms or a combination of them was always present. Certain other noteworthy differences will be pointed out in the symptomatology after the analysis of these cases is gone into.

The material from which this analysis is made differs not at all from the material which supplied the basis for the observation in the cases of Traumatic Neurosis.

The Observation.—There were seventy-three cases diagnosed as traumatic hysteria. The average age of these patients was thirty-six and one-half years. Fifty-four were married, fourteen single and five widowed. The large proportion of married folk who develop functional nervous diseases as a reaction to injuries was commented upon in the preceding paper. The economic difficulties which the married individuals experience to a greater degree than their more fortunate single fellows, are undoubtedly a predisposing factor in the precipitation of these conditions. Regardless of other factors which may be exerting their influence, the single factor of added stress which the married individual experiences simply because he must look after a family, plays a great part in bringing about functional nervous symptoms. Besides this economic factor, married individuals are predisposed because of their civil condition to difficulties in the adjustment of their sexual and other in-

¹ Page 336 of this issue.

instincts which single individuals are not so likely to experience. In the cases of traumatic neurosis the only psychological factor at work seemed to be the emotion of fear and the instinct to flee from an unpleasant situation. In too many of these cases of traumatic hysteria to make it unimportant, difficulties were found with the handling of other instincts and emotions, particularly in the sexual life. These matters will be brought out later in the discussion of the individual cases.

In these cases of traumatic hysteria too, there was almost invariably found a previous instance of some mental or physical abnormality. This was not true in the cases of traumatic neurosis. Despite the fact that very few of these individuals admitted having been nervous before the accident, careful questioning brought out in the history undoubted signs of important physical and mental difficulties. Thirty-three of these patients had varying periods of unconsciousness, a history of which was unobtainable in forty. It will be recalled that in the cases of traumatic neurosis the majority of the patients gave a history of having been unconscious. The onset of the original symptoms complained of in these cases of hysteria was sudden in 56; and in the remainder the average period of incubation, as it might be called, following the date of injury, was ten days. In this respect the averages are the same as in the cases of traumatic neurosis. Regarding the symptomatology, however, there is a marked difference. Headaches were complained of in only 32 cases out of 73, whereas in the cases of traumatic neurosis, 83 patients made this complaint out of a total of 102. Dizziness was complained of in 19 cases only as against 80 in the latter condition. The nature and severity of the injury was apparently unimportant in deciding the type of reaction. There were head injuries in 54 cases and back injuries in 13. In the remainder of the cases the injury was to one of the extremities. The blood pressure readings averaged 125 systolic and 85 diastolic. While it is true that the onset of

the symptoms was sudden in many cases, it was invariably found that many months passed before the total maximum disability was reached. It was almost invariably true that while one or two symptoms were complained of almost immediately following the accident, these became much worse gradually and other symptoms were added as time went on, until finally the maximum disability was reached at or just before the time arrived for a consideration of these cases by the tribunal by whom the award was to be made. In the 21 cases which we were able to follow up after the litigation was ended, the history was invariably obtained in all except one case that the disability gradually receded. One patient did not resume work until nine months after the award was made. In one case where this gradual reduction of disability did not occur, recovery from the symptoms complained of took place within an hour after the award was made. It was true also in all of these 21 cases that while the actual disabling symptom or group of symptoms disappeared, the patients persisted in a number of subjective complaints and insisted that they were not entirely well. In other words the symptoms were relieved, but the hysterical character was, of course, not changed.

Another point of difference is that in the cases of traumatic neurosis it was possible to improve a great many of the patients by treatment and a small number even recovered and went back to work before their cases were called up for settlement. At no time, however, have I been able to get any noteworthy improvement or effect a cure in any case of traumatic hysteria before the final adjustment of the litigation was arrived at. Besides this, I have never seen a case that began with the symptoms grouped under the term traumatic neurosis, develop at any time any of the symptoms grouped under the term traumatic hysteria. Almost invariably, too, it is possible to find in the actual symptom complex presented by the hysterical patient a clew to the mechanisms operating to produce

the disability. In traumatic neurosis, however, the symptoms complained of were almost constantly the same in each case, and one is led to believe by this fact alone that the former group of cases is probably based on a real anatomical and physiological pathology, whereas the hysterical group is probably almost entirely psychologically determined.

Another observation of great importance is the fact that in my cases of traumatic hysteria, I became impressed with the difference in the mentality and intellectuality of these patients as compared with the patients constituting the traumatic neurosis group. The latter are more like average normal individuals who have been tired and worried and frightened. The hysterics, however, were essentially abnormal personalities. They showed great emotional instability. Most of them were definitely asocial; many showed mental retardation up to definite degrees of feeble-mindedness; many were shy, reserved and sensitive from early childhood, showing great inability to adjust themselves to the ordinary stresses and experiences of life. They were quarrelsome, suspicious and selfish. One became convinced quickly of the genuineness of the complaints of the patients who suffered from traumatic neurosis. The contrary impression was received from the patients who were grouped under the heading of traumatic hysteria. One listened to their complaints with a feeling of their insincerity, and then the suspicion often came uninvited to one's mind that these patients were deliberately misrepresenting. This does not apply to all cases by any means. In a great many of these patients the mechanisms at work were so close to their conscious thinking, that one had difficulty in differentiating them from true malingerers. It was just in this type of case that the greatest degree of mental enfeeblement was found. It seems impossible to escape the conclusions that in nearly all the cases of traumatic hysteria, the desire to obtain an advantageous settlement for the injury motivates the

symptom complex presented. Often this central nucleus in the psychological constellation is obscured somewhat by other difficulties. The instinct of acquisition and the opportunity to play, for once, a powerful rôle and focus the attention of one's fellows on oneself, undoubtedly helps to produce the symptoms, although these factors are reinforced by compromises with other instinctive tendencies. The most frequent maladaptations to instinct of this later type are those of sex. As examples the following cases may be instructive:

This patient was a Pole, forty years of age, who had been married for twenty-five years. There were no children. The wife was a robust, comely Polish woman, of a very virile and dominant type. She had always made the decisions for the family and imposed her will without resistance from the husband on all occasions. For many months before the injury the patient had been weak sexually, "no good," as she expressed it. On July 27, 1918, a plank carried in the hands of some fellow workmen struck him in the back while he was in a stooping posture. He immediately fell to the floor unable to move his lower extremities and was taken home in an automobile. Thereafter he suffered from a particularly persistent astasia-abasia. He complained bitterly of tenderness and pain in the spine and back and was treated for many months for a possible injury to the spine. It was only after he was examined by a competent orthopedist and many x-ray examinations were made that a neurologist was called in, hysteria being suspected. At the time of the examination extensive functional anesthasias were found, but no organic neurological sign was discovered. There was no disturbance of the bladder or rectum and the patient complained at the time that I saw him, eleven months after the injury, that he had completely lost sexual power since the accident. He feared that his sexual power had been completely destroyed forever. No treatment ever did him any good.

One can readily see that while the instinct of acquisition played necessarily a minor rôle in this case, it being overshadowed in importance by the sexual difficulties mentioned, nevertheless it was the central nucleus, the heart of the trouble, for only by ascribing his physico-sexual debility to the injury could he justify the illness, and only by being paid well could he propitiate his spouse and reassume any importance in her eyes. The instinctive maladaptations here are those which this patient showed to sex, the instinct of acquisition² and the instinct of positive self-feeling.

This man was the usual dull, stupid, uninteresting type, lacking in education and possessed naturally of poor mental stuff. Another interesting observation in this case was the fact that during his stay in the hospital he brightened up considerably and was on a fair road to recovery apparently during the first week when we succeeded in keeping his wife away, but he suddenly relapsed after her visit and never improved. She saw him daily after her first visit.

Another patient was an individual of feminine make-up physically, whose voice and attitude of mind were very suggestive of womanly qualities. He suffered from *astasia-dysbasia* and assumed an attitude suggestive of the "hunch back" at all times. Every possible method of examination was made to exclude organic disease or injury to the spine, and his genuineness being under suspicion, he was followed by detectives for weeks, but was never found in any excepting the above described attitude. One of his most troublesome symptoms was his irritability towards his wife. They had been married for nine years and had one child eight years old. His wife gave the history that for several years the sexual act had become distasteful to him, and that he had left her on several occasions and had maltreated her. Since the accident the patient complained that he has had complete sexual impotence.

This man's physical abnormality consisted of a feminine distribution of fat and hair and general physical make-up, and an infantile development of the sexual organs.

It can readily be seen from these two cases that aside from the possibility of gaining something material from the accident which they had sustained, an excuse was offered them to avoid adjustment of sexual matters which were either distasteful or impossible for them. It is not an unimportant coincidence that these two cases were the most troublesome and most obstinate of the series. In the following cases dissatisfaction with the job undoubtedly motivated the symptoms to a considerable degree.

This patient, thirty-seven years of age, married for five years, with no children, developed a hysterical palsy of the right upper extremity after he had been carrying a heavy picture from the store where he was employed for a distance of about one mile. He had carried similar frames many times before without trouble, but on the day of the onset of his symptoms, after he had delivered the frame he found that he could not move his right upper extremity and complained of pins and needles and burning in the right shoulder, hand and arm. Examination readily excluded any organic disease or injury. There was complete anesthesia of functional type in the affected extremity. The history brought out the fact that this patient considered himself a high-class artisan, and in his country of origin he had been a man of some importance, besides being a painter of talent. Since coming to this country he had to do what he looked on as unworthy work; particularly did he object to carrying his work to prospective customers. On the day that the palsy was first noticed he felt bitterly resentful at what he considered an indignity. No amount of reassurance concerning the non-organic nature of his condition ever succeeded in improving him much, and six months after the onset of the trouble he was still incapacitated. This man was considerably under weight

² MacDougall's *Social Psychology*, page 322.

and somewhat anemic, but otherwise physically well. Mentally he was acutely sensitive, resentful and quite irritable.

The next case illustrating this point is that of a young Irishman whose age was twenty-four. He was single and had been in this country only five years. In Ireland the father was a land-owner, quite well-to-do, and the boy was a student in an Irish university. He came to America after a quarrel with his parents and lived with a brother in this city. He was forced to go to work, and made a rather precarious living. The life here was very distasteful to him. He made no serious whole-souled efforts, was quite depressed, and for some time before the accident had been very unhappy. The injury consisted of a fall of about ten feet without loss of consciousness. There was no evidence of his having injured either the vault or the base of the skull. They were about to discharge him from St. Vincent's Hospital, when he became very hysterical, had a convulsive attack, and suffered from astasia-abasia. He was seen a number of times by several very competent neurologists and all agreed that he was a case of traumatic hysteria. In discussing his case the patient's brother refused to allow a final settlement, but both the patient and the brother agreed to accept enough money to enable him to return to Ireland and live comfortably. As soon as this arrangement was made the patient began to improve rapidly, and whereas he had not stood alone for many months, he was able to go down to the steamer and sail for Ireland. I have since been reliably informed that the boy has shown very great improvement and that he is now living with his mother and father. The brother here tells me that the case was settled for the sum necessary to cover the expenses of the boy's trip to his home in Ireland.

In both these inadaptably individuals the self-regarding sentiment, as MacDougall would call it, was being injured by their enforced stay in an unpleasant environment. The injury simply called

into play the instinct of acquisition to enable them to escape the difficulty.

Many of these patients suffered an actual organic injury which may serve as a basis for the hysterical manifestations which followed. This was true in almost two-thirds of the cases. For instance, one man, an Italian, married, suffered a fracture of the lower end of the humerus. This fracture did not involve the elbow-joint. X-ray and physical examinations failed to make out any injury to that joint, yet the extremity was held in an awkward attitude and the joint could be moved only by overcoming a great deal of resistance. Passive motion when done in this way, however, resulted in complete mobility of the joint. This contractural palsy relaxed during sleep, and the patient's extremity was moved freely during experimental anesthesia. In this patient the intense desire to return to Italy and to see his father and mother, played an important part in the continuance of his hysterical syndrome. His dreams were frequently of the village in Italy where he had lived with them and he volunteered the statement that he often heard his parents calling him home in his dreams.

Another patient who well exemplifies the building of an hysteria based on an organic injury is that of a young American aged twenty-four, who was overcome by carbon monoxide poisoning on July 3, 1918. He suffered from an hysterical aphonia, could not even whistle or sing or make any sound for many months. About one year after this he fell off a ladder and fractured the bones of the right forearm. He began to speak immediately, but when the injured extremity was taken out of the cast he developed a hysterical palsy.

The case of a woman who developed serratus magnus palsy following an operation to correct a bony displacement resulting from an injury, is another case in point. This patient not only had a bad ankle joint, but had a traumatic neuritis of the nerve of Bell subsequent to operation, which was verified by electrical reactions,

the presence of winging of the scapula, etc. Following this she developed a complete hemi-anesthesia of the hysterical type.

A young Italian who sustained a fracture of the middle third of the femur with a good result and less than one half-inch of shortening, nevertheless developed rather slowly thereafter an hysterical palsy of the injured extremity which could not be influenced by any sort of treatment.

It has been borne in on me frequently that the psychological determinants in certain cases of hysteria rise close to consciousness. Certainly many of these cases have their difficulties so close to the surface that the suspicion of malingering is often justified.

An illustration of this point is that of a negro who fell, injuring his head. There was no depression of the skull and no fracture visible on x-ray examination, but the patient was unconscious for a short period after the accident and bled from the right ear a few hours. He was able to go home after a week's stay in the hospital, but gradually thereafter developed many complaints, the chief of which was an unsteady gait and an apparent confusion with mental retardation. When I saw him many months after the injury a complete neurological examination was negative excepting for certain peculiar disturbances of gait and station. Injury to the internal ear was excluded by Hitzig's and Barany's caloric tests. Although he walked without staggering or swaying by the aid of a cane or on his brother's arm, when walking alone he staggered and swayed in all directions and when standing alone with the eyes either open or closed, he would sway and at times almost fall, always however, in the direction of the person close to him. When his brother was standing by, efforts were always made by the relative, to grasp him and prevent him from falling. Occasionally the patient fell towards me, but when his brother was not present during the examination, this symptom was not nearly so pronounced, because I made no efforts to prevent him from falling. He

merely swayed unsteadily and never in the same direction twice. Numerous Barany and Hitzig's tests were made, but nothing abnormal was found. There were no errors of pass-pointing and no spontaneous nystagmus. At first I thought the patient was deliberately malingering and asked for a two weeks' observation before I could make up my mind. At the hospital where he was placed for observation, he remained in bed practically all the time and continued to stagger and sway on the few occasions that he got up. He developed a mutism and a negativism which was quite pronounced. He refused food and would only accept it when the nurses fed him with a spoon. For two whole days the nurses were instructed to leave the trays by his bed and walk away. The food was not always taken by the patient, although on two occasions he did eat. Occasionally he would break out from his mutism in sharp protest, declaring that he wanted to go home; but aside from these occasional outbursts he said nothing. If an examination was attempted during his stay in the hospital, he immediately went into a rigid state and nothing could be done with him. On a number of occasions he was tested both by the house staff and myself for station and gait, but the swaying and staggering were always present. At the hospital too he developed for the first time a complete hemi-anesthesia to all qualities of sensation except vibration and muscle-tendon sense. The anesthesia was undoubtedly functional. When the patient was discharged, a suspicion had arisen as to the genuineness of the case, which persists even now. The negro was of very low mentality. The negativism and mutism were not part of a dementia precox, but were undoubtedly functional.

A particularly interesting case where the suspicion of malingering occurred was that of a woman aged forty-four, who had been arrested for stealing several thousand dollars' worth of dresses from a large department store in this city. The trauma in her case had consisted of a fall during a

fainting spell several months before the onset of the symptoms. She began at first with strange periods of amnesia and confusion. At the table or in the parlor when the family was present, she would suddenly lose touch with what was going on, did not answer questions and stared into vacancy and would utter irrelevant phrases such as "where is he, ain't it awful, what has he done," etc. These periods of amnesia would last for a half hour or so and then the patient would go on as if nothing had happened. Occasionally she would faint. It seems that for some time she had been stealing from her place of employment dresses of gaudy colors and of the type that a gay girl of eighteen or nineteen would wear. The family thought that she was buying these things. When she was arrested a number of trunks were found filled with gaudy dresses, fancy shoes, sweaters, bathing suits, beautiful hats suitable only for young women and other trinkets such as a staid woman of forty-four would have no use for. None of these things had ever been disposed of. There seemed to be no motive for the robberies. The patient came from a good family, had an independent income and did not work because of necessity, therefore it was positively determined that the robberies were not for gain of any kind. She never wore any of the things nor did she dispose of them in any way. When her house was searched all the missing things were found, some in boxes unopened. The judges who tried her case became convinced that there was something mentally wrong with the patient and turned her over to several psychiatrists for study. The history, which was carefully gone into, showed that she was always rather sensitive and childlike and shy. She was cold and unresponsive to her family except to her mother, whom she idolized and waited on continuously. She loved the children of her sisters excessively and liked to have them about her and often bought them expensive presents. She said that she began taking the skirts and dresses, bathing suits, etc.,

without giving the matter any thought. She seemed to obey an impulse. She did not think of the morality of the situation at all, but would bring the trinkets home and put them on and then store them away in trunks. She herself always wore black and dressed very sedately. She had stolen many packages of rouge and powder, but personally never used any of it. She was extremely religious, but even now it does not seem to her that what she did was stealing.

About the time that the stealing first began, she met a man of about fifty-six in a rather questionable way. It was while they were both in a public conveyance. The man was very kind to her, and seemed to influence her against her will she said. She met him a number of times and was taken to road houses and to various fashionable restaurants, but so far as we could learn the flirtation went no further. This was the first love affair the patient had ever had. She admitted that she was very fond of the man and was extremely happy at the thought of being his wife. The proposal of marriage, however, never came, and the man seemed content with taking her to gay places where young people were wont to go. The patient admitted that she often desired the return of her youth and that she envied the young people whom she saw enjoying themselves, particularly when she listened to her companion express his admiration for them. He said on one occasion, "If you and I were younger I would really think of talking marriage to you." It was about this time she began to take the things for which she was later arrested. On talking the matter over with the psychiatrists whom they had requested to examine her, the judges finally decided to send her to a sanitarium for three or four months and not to a jail. She began to get well quickly and her amnesia attacks disappeared. The only treatment aside from the routine sanitarium care was that it was insisted upon that her affair with the man be broken off.

The injury from which this patient dated

her troubles played, of course, a very minor part in the production of the symptoms; yet it enabled her to fasten her difficulties on something outside of herself and in this way spare her self-regarding sentiment. It would seem that the maternal instinct and the desire to gratify her undeveloped sex instincts, were also important factors in the production of the hysteria. It is not difficult to see how the possibility of malin-gering in a case of this kind could very well arise.

CONCLUSIONS

1. The functional nervous reactions to injury are divided in two groups, possessing differentiating characteristics.

a. In traumatic neurosis the reac-

tion is apparently caused by fear with immediate injury to the nervous system by actual trauma or later injury by sleeplessness, restlessness and a general physico-psychical depression. Most of these individuals are essentially normal, the injury being almost entirely responsible.

b. In hysteria, the reaction to the injury is probably not determined by any primary or secondary pathological considerations directly due to the injury, but is entirely an instinctive maladjustment in individuals who are both physically and mentally inferior.

2. A carefully analyzed case of traumatic neurosis will not show any of the true hysterical stigmata.

CEREBRAL GLIOMA AND ACUTE HEMORRHAGIC ENCEPHALOMYELITIS

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The record of this case is interesting from a number of standpoints. The ante-mortem diagnosis of epidemic encephalitis was not entirely borne out at autopsy, but a hemorrhagic encephalomyelitis and a glioma of the right cerebral hemisphere were found. The existence of the glioma, though it was of large size, had never been suspected, as it had given neither general symptoms nor focal signs.

The question of whether hemorrhagic encephalitis may not be a form or stage of epidemic encephalitis is asked, but cannot be answered in my opinion before our knowledge of both conditions is more definite than it is at present.

HISTORY.—Patient, B. R., aged fifty-one, born in Germany. Admitted to the hospital June 7, 1919.

Chief Complaint.—Headache for three weeks.

Family History.—Father died at forty-nine of pulmonary tuberculosis. Mother died at sixty-seven of bronchitis. Two brothers died in childhood, causes unknown to patient. Four brothers and four sisters living and well. Grandmother, uncle and two aunts on the maternal side died of cancer. No history of diabetes, renal or cardiac disease in the immediate family.

Past History.—General health fair. Measles, mumps, chicken-pox in childhood. No typhoid, no acute rheumatic fever. Influenza and pneumonia six months ago followed by considerable prostration. No operations. Frequent headaches in childhood, but does not recall any details. Occasional headaches previous to present illness.

Eyes.—Glasses have been worn for the past four years for presbyopia. There is no blurring of vision.

Nose.—Occasional rhinitis.

Pharynx and Tonsils.—Frequent sore throat and tonsillitis in childhood.

Respiratory.—Negative.

Cardiac.—Occasional palpitation when excited and after undue exertion. No dyspnea.

Gastro-intestinal.—Appetite good; bowels constipated, requiring daily catharsis.

Genito-urinary.—Urination three to four times a day. No nocturia.

Menses.—Periods began at twenty-one, lasted two to three days with three or four weeks between periods. The intervals were not regular. Periods stopped three years ago. No discharge since menopause.

Nerves.—Always nervous and high-strung. Easily upset by trifles.

Personal History.—Born in Germany, has lived in the United States for twenty-six years. Is widowed, her husband having died two years ago. She has one son. No history of miscarriage. She does light housework around her home and helps in her sister's hair parlor. Eats and sleeps regularly. Takes one cup of tea and three cups of coffee daily. Alcohol: An occasional drink, but no habit.

Weight.—Best weight was 136 pounds, three years ago. Average weight is 120 pounds. The present weight is 110 pounds.

Present Illness.—The patient is in a rather stupid mental state, her answers to questions being slow and indefinite. The following history was obtained from her sister.

Three weeks ago the patient was found unconscious, rigid and frothing at the mouth. The unconsciousness lasted about an hour. She stayed in bed for a week, complaining of weakness and tingling in her left leg and arm and marked drowsiness. The right corner of her mouth was

drawn over. Since the onset of this illness she has had constant headaches, not localized in any portion of the head. Any exertion seemed to increase their severity. There was no nausea or vomiting, no spots or flashes before the eyes. After one week in bed she got up and during the past two weeks has been about, but was always drowsy, dropping off to sleep very easily. There has been no evident paralysis, the weakness in the left side having cleared up.

PHYSICAL EXAMINATION

General Appearance is that of a middle-aged woman resting comfortably in bed, nervously exhausted. There is no dyspnea, no cyanosis.

Skin is negative.

Mucosa.—No pallor.

Head is symmetrical. Hair normal in amount and distribution. Sinuses: Slight mastoid tenderness on deep pressure. Eyes: Slight exophthalmos; no Stellwag, no Möbius. Pupils are equal, central, react to light and accommodation. Both eyelids are nearly closed. The patient is able to open the right eyelid fully, the left to about half the normal extent. Ears: No discharge. Drums appear normal. Nose: No discharge. Normal to external examination. Mouth: Tongue is coated. Upper teeth are false. Gums are retracted; there is marked pyorrhea. Pharynx is congested. There is flattening of the left side of the face.

Neck.—No rigidity.

Superficial Lymph Nodes.—No pathological enlargement.

Spine.—Spinous processes of the lumbar vertebræ are unduly prominent.

Thorax is symmetrical; apparently normal.

Breasts are flat; no masses.

Lungs are normal.

Heart.—Apex impulse is neither visible nor palpable. No thrills; no murmurs. Aortic second sound is slightly louder than the pulmonic second. Pulse is equal, regular in rate, rhythm and force. Tension is

normal. Cardiac outline is normal, left limit of dullness being 9 cm. in the fifth interspace. Blood pressure 110-63.

Abdomen is soft; no masses, tenderness or rigidity. Liver and spleen not felt.

Kidneys.—Lower pole of the right kidney palpable on deep inspiration.

Pelvic and rectal examinations not made.

Extremities.—No edema or tremor. Bilateral hallux valgus.

Weight.—100 pounds.

LABORATORY EXAMINATIONS

<i>6/8/19 Blood Count:</i>	
Red blood cells.....	5,660,000
White blood cells.....	9,000
Hemoglobin.....	70%
Polymorphonuclear leucocytes.....	74%
Lymphocytes.....	20%
Transitionals.....	2%
Large mononuclears.....	3%
Eosinophiles.....	1%
<i>Blood Urea:</i> .29 gram per liter	
<i>6/9/19. Stool for routine examination:</i>	
Small, pasty stool	
Color: Yellowish brown	
No blood	
No ova or parasites	
A few undigested meat fibres	
<i>6/9/19. Blood Count:</i>	
Red blood cells.....	5,580,000
Hemoglobin.....	70%
<i>6/10/19. Blood Wassermann:</i>	
Alcohol antigen.....	negative
Cholesterin antigen.....	negative
<i>6/10/19. Lumbar Puncture:</i>	
Approximately 15 c.c. of clear fluid removed under very much increased pressure. The first 8 to 10 c.c., spurted out and ran into the test tube in a continuous stream.	
Cell count.....	6 lymphocytes
Globulin.....	Negative
Wassermann.....	2 c.c. 1 c.c. 6 c.c. 2 c.c.
Alcohol.....	Neg. Neg. Neg. Neg.
Cholesterin.....	Neg. Neg. Neg. Neg.
Culture.....	Sterile

NEUROLOGICAL EXAMINATION 6/11/19

VOLUNTARY MOTOR SYSTEM. Abnormal Attitudes and Deformities.—There is no deformity of the spine, abnormal abdominal protrusion or abnormal position of the head. There is marked hallux valgus on each side. There is a catatonic rigidity on the left side; the arm, and to a less extent the leg, will be held for several minutes in any position in which it is placed.

Gait.—The patient is unable to stand or walk.

Coördination.—Equilibratory not tested, as patient is unable to stand. Non-equilibratory: Finger-to-nose, pointing and pass pointing, heel-to-knee and toe-to-object-above-recumbent-patient tests are normal on the right side, but are performed with only moderate precision on the left. There is no dysmetria or adiadochokinesis.

Skilled Acts.—There is no dyspraxia. Speech is slow and deliberate, but there is no dysarthria or aphasia.

<i>Reflexes:</i>		
<i>Deep</i>		
	<i>Right</i>	<i>Left</i>
Jaw.....	Present	Present
Pectoral.....	Present	Present
Biceps.....	2	3
Triceps.....	2	3
Radial.....	2	3
Ulnar.....	2	3
Wrist.....	2	3
Suprapatellar.....	Absent	Present
Patellar.....	2	3
Achilles.....	2	3

<i>Superficial</i>		
Supra-umbilical.....	Present	Absent
Suprapubic.....	Present	Absent
Upper lateral abdominal.....	Present	Absent
Lower lateral abdominal.....	Present	Absent
Plantar flexion.....	Present	Absent
Babinski.....	Absent	Present

1 = slight; 2 = active; 3 = very active; 4 = with transient clonus; 5 = with permanent clonus.

Abnormal Involuntary Movements.—None present.

Muscle Strength.—The movements of the neck and shoulders seem equal on the two sides. All movements of the left arm, forearm, hand and fingers, as well as the left thigh, leg and foot, are perceptibly weaker than on the right side.

Muscle Status.—Consistency is normal; contour, except for a flattening of the left side of the lower face, is normal.

Abnormal Associated Movements.—The pronation sign of Strümpell, the automatic pronation sign of Babinski, the mechanical grip, the thumb sign of Klippel-Weil, the interossei phenomenon of Souques, the signs of Neri with the patient recumbent, and the sign of Marie-Foix are all absent. The sign of Bechterew and the trunk-thigh sign of Babinski are present.

Nerve Status.—Nothing noted.

General Sensory.—(Examination not entirely satisfactory because of the patient's mental condition.) Touch: Acuity, localization and discrimination seem normal. Pain, temperature (cool and warm), vibratory, pressure, muscle tendon sense and stereognostic perception are normal.

Cranial Nerves and Head. *Olfactory and Nose.*—The sense of smell is normal.

Optic and Oculomotor Apparatus.—Visual acuity not tested. Perimetry, roughly tested, normal. There is no photophobia. The pupils are in mid-dilatation, the left is slightly larger than the right. Both are regular. The reaction to light is sluggish on both sides, more especially on the left. The reaction to accommodation seems normal. There is bilateral ptosis. With considerable effort and elevation of the eyebrows the patient is able almost completely to open the right eye. The left one can be opened only about half of the normal extent. There is no strabismus. The conjugated eye movements seem normal. Nystagmus is absent. Fundi: The veins appear congested in each eye. The disks are normal and there are no hemorrhages.

Acoustic Nerve and Ear.—A watch is heard 20 inches from each ear. Air conduction is greater than bone on each side.

Trigeminal Nerve and Mouth.—The movements of the jaw in all directions are normal. Sensation is normal.

Facial Nerve and Face.—Right side normal. On the left side there is a marked flattening and weakness of the lower face.

Glossopharyngeus and Vagus Nerves, Pharynx and Larynx.—The position of the uvula is medial. Palatal and pharyngeal reflexes are normal. Swallowing, respiration and cardiac actions are normal.

Spinal Accessory Nerve.—There is no evidence of weakness of either sternomastoid or trapezius.

Hypoglossus Nerve and Tongue.—The tongue is protruded in the mid-line and its movements in all directions are equal. Articulation is normal.

CLINICAL EXAMINATIONS AND NOTES

6/10/19. *Examination of Eye Grounds.*—Discs are clearly outlined. Blood vessels are not tortuous. No hemorrhages. No retinitis.

6/11/19. When the patient presented herself for examination to enter the hospital her complaints were so general and vague and her general physical examination so nearly negative, that the admitting physician did not believe it necessary to have her enter the hospital. During the first three days of her stay in the hospital she was listless and inattentive, answering questions slowly and indefinitely. Her only complaint was a slight headache. After the lumbar puncture yesterday her condition became rapidly worse, the lethargy becoming stupor and the signs of a left hemiplegia developing.

At present she does not reply to questions but will protrude her tongue and move her arm when asked to do so. She winces when pinched or when the muscles are squeezed. Pupils are in mid-dilatation, the left greater than the right. The right pupil reacts very sluggishly to light and the left not at all. The pulse is slow and regular. Blood pressure 145-85. Biceps and patellar reflexes are present on the left. Urination involuntary to-day.

6/13/19. Patient is still very somnolent but can be roused. She appears to understand questions and answers them intelligently but lapses back into sleep at once. The right side of the forehead is more wrinkled than the left. The right corner of the mouth is drawn over. The left side of the face is flattened. When requested to open her eyes she fails to do so, although she undoubtedly understands the request and endeavors to comply, as shown by elevation of the eyebrows. She is able to protrude the tongue. The lower jaw does not sag. The pupils are moderately contracted and react sluggishly to light. There is no strabismus. There is no difficulty in swallowing. Involuntary urination continues.

6/14/19. Patient is more somnolent than yesterday; cannot be roused. Swallows with difficulty. The right pupil is larger than the left. There is no strabismus. The reaction to light is very slight, merely a quiver of the iris.

6/16/19. The patient has continued about the same since the last note until 7.45 this evening when the condition suddenly became worse, growing markedly dyspneic and cyanotic, respiration ceasing after a few minutes.

During the nine days of observation in the hospital the temperature ranged from 98° to 100°, the pulse from 65 to 85.

AUTOPSY

June 17, 1919. 13 hrs. P.M. By Dr. R. F. E. Stier.

Anatomical Diagnosis

Glioma of brain
Cerebral hemorrhages
Chronic glomerulonephritis (vascular)
Arteriosclerosis
Atheroma of aorta (moderate)
Edema and congestion of lungs
Chronic endocarditis (mitral)
Cloudy swelling of liver
Fatty infiltration of liver (slight)
Acute splenic tumor
Congestion of pancreas and kidneys
Polyp of uterus
Chronic myocarditis
Museum specimen: Brain and cord.

The body is that of a white female measuring 164 cm. in length. There is an abundant amount of light brown hair. The eyes are in mid-dilatation, pupils equal. The cornea are glassy and the conjunctivæ are of a pale yellow color. The upper teeth are replaced by bridgework and a false set. The lower jaw shows a marked pyorrhea. The mucous membranes of the mouth are pale. The nasal septum is intact and shows a small amount of bloody discharge. The ears are negative. Over the neck and back there is post mortem lividity. The upper extremities show emaciation and are rigid. The breasts are well developed and contain a large amount of

glandular tissue. No masses palpated. Over the thigh and shin there are a few varicose veins.

Brain.—The convolutions appear swollen, slightly flattened. The pia mater shows a marked engorgement. The section of the pons shows in the left lateral mass of the pons, near the left middle cerebral peduncle, a hemorrhagic area which mea-

numerous congested areas. The right cerebral hemisphere appears larger than the left and in the median line bulges and makes a deep impression into the mesial surface of the left lobe. As a result the mesial surface of the left lobe is very convex. Section through the frontal lobe near the anterior pole shows rather a circumscribed hemorrhagic area 2.5 cm. in

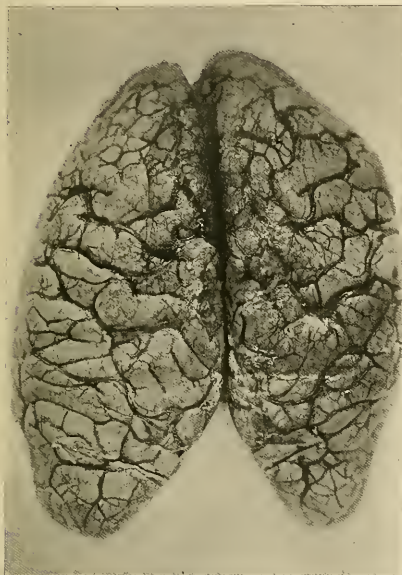


FIG. 1. CEREBRUM SHOWING ENLARGEMENT OF RIGHT HEMISPHERE, FLATTENED CONVOLUTIONS AND CONGESTION OF SURFACE VESSELS.



FIG. 2. VERTICAL SECTIONS OF CEREBRAL HEMI-SPHERES SHOWING TUMOR AND HEMORRHAGES.

sures 2 cm. by 8 cm. In this area the brain tissue is absent and is replaced by blood clot. Other sections through the lower portion of the pons and upper portion of the medulla show several petechial hemorrhages. In the floor of the left ventricle beneath the stria acustica there is a small petechial hemorrhage and the capillaries are congested. In the anterior portion of the dentate nucleus of the cerebellum there are several small petechial hemorrhages. The white matter of the cerebellum shows

diameter. This area is gelatinous, translucent, and there is definite encephalomalacia. Sections taken a cm. apart show this hemorrhagic and necrotic area involving practically the entire central portion of the right cerebral hemisphere. It becomes larger, more hemorrhagic, as the mid-portion of the brain is reached. It involves the external capsule, the lenticular nucleus, the internal capsule and the caudate nucleus. Also near the mid-portion of the brain there is a similar large hemor-

rhagic softened area in the mid-frontal convolution which involves the mid-radiation of the corpus callosum. This area is of a yellowish brown translucent necrotic substance in which are found gelatinous masses.

Abdomen.—On opening the abdominal cavity the panniculus measures 4 cm. It is of a golden yellow color. The intestines lie free. The cavity contains no fluid. The appendix measures 6

slightly increased in amount. The pericardium is smooth throughout showing a few areas of thickening. On opening the heart the muscle is of a deep reddish-brown color showing a few fibrous streaks. The tricuspid and pulmonary valves show no change. In the mitral leaflet there are a few fatty plaques. The free margin is thickened and puckered. The chordæ tendinæ are slightly thickened and shortened. The aortic valve shows a slight thickening.

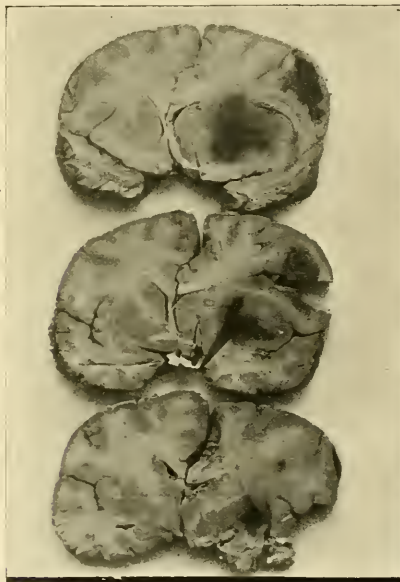


FIG. 3. VERTICAL SECTIONS OF CEREBRAL HEMI-SPHERES SHOWING TUMOR AND HEMORRHAGES.

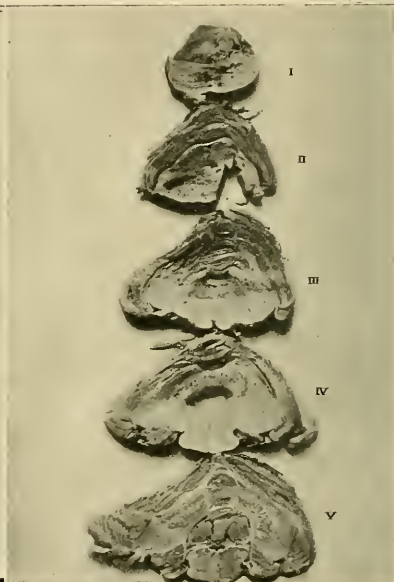


FIG. 4. SECTIONS OF BRAIN STEM. MULTIPLE HEMORRHAGES.

cm. in length and its apex shows congestion. The mucosa of the intestines is shining; several coils of the ileum are adherent to the peritoneum at the level of the last lumbar vertebra. The liver reaches to the free costal margin. The bladder is distended and reaches about 3 cm. above the symphysis.

Thorax.—On opening the thoracic cavity the lungs fill the cavity and show only a few adhesions which are broken quite readily. The pericardial cavity contains a small amount of clear straw-colored fluid.

Heart weighs 250 grams. The epicardial fat is

Measurements		
Tricuspid valve	11	cm. in circumference
Pulmonary valve	7.5	cm. in circumference
Mitral valve	9	cm. in circumference
Aortic	7	cm. in circumference
Left ventricle	10	mm. in thickness
Right ventricle	4	cm. in thickness

Aorta.—It is diminished in elasticity and throughout its entire course shows a few fatty plaques.

Right Lung weighs 300 grams. The pleura over the entire lung is smooth except at the apex where it shows areas of thickening and a

few fibrous tags. The upper lobes are air containing, the lower lobe shows an increased resistance. The pulmonary vessels are patent. The bronchi are congested and on pressure exude a frothy bloody fluid. On section the upper lobe is of a salmon color in its anterior portion while the posterior portion of the lung shows a red color. The lower lobe is of a reddish pink color. It is markedly congested. There is a small amount of emphysema.

Liver weighs 1150 grams. The capsule is smooth and shining. Below the capsule are whitish areas and a few circumscribed golden colored areas. On section there is a more or less homogeneous, glossy, chocolate colored appearance. The lobules are not evident, and in areas, particularly situated around the portal spaces, are lighter yellowish areas.

Gall Bladder.—It is distended and contains a liquid greenish colored bile; also numerous

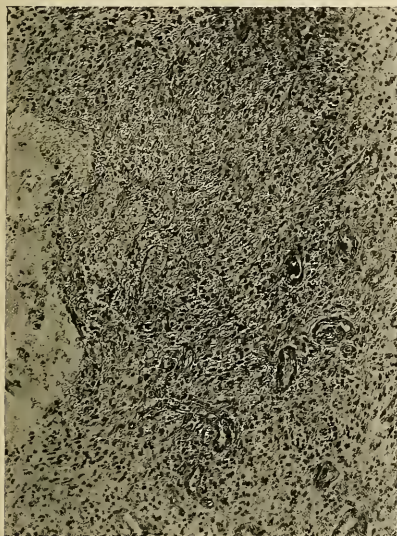


FIG. 5. SECTION OF GLIOMA SHOWING CHARACTER OF TUMOR, INTIMAL PROLIFERATION OF SOME OF THE VESSELS, AND NECROTIC AREA RESULTING FROM AN OLD HEMORRHAGE.



FIG. 6. A PORTION OF TUMOR SHOWING RECENT HEMORRHAGES.

Left Lung weighs 200 grams. The pleura over the entire lung is smooth and glistening except at the apex where it shows a slight thickening. The upper lobe is air-containing and crepitates. The lower lobe is apparently not air-containing and distended. The pulmonary vessels are patent and contain a large amount of fluid blood. The bronchi are congested and exude a frothy fluid. On section the upper lobe is of a salmon color and shows congestion, which is more marked in the dependent portions. Shows a slight amount of emphysema. The lower lobe is of a deep red color. The blood vessels are markedly congested.

faceted gall stones of various size which are of an olive color, the center of which are white. In the gall bladder are many yellowish flakes. The mucosa of the gall bladder is roughened.

Spleen weighs 100 grams. The capsule is of slate color showing areas of thickening. It is slightly wrinkled. At its upper poles is a deep fissure and an accessory lobe. On section the spleen is very friable. It is of a mottled appearance showing deep red and lighter areas. The Malpighian corpuscles are not very evident. There is a marked increase in the content of the pulp.

Kidneys weigh 110 grams each. The capsule

strips quite readily, leaving a smooth cortex which is of a deep red color. The stellate veins are quite prominent. On section the kidney is of a deep red color. The differentiation between cortex and medulla is quite evident. The arcuate arteries stand out quite prominently. The Malpighian corpuscles stand out as whitish pinpoint areas. The pelvic fat is about normal in amount and the renal vessels show no change.

Pancreas.—It is of a reddish color. Shows a

os. The uterine muscle shows a congestion, otherwise nothing remarkable.

Ovaries and Tubes.—The tubes show a congestion and the ovaries show some old and more recent corpora lutea.

Rectum.—Apparently unaltered.

Stomach.—Contains some undigested food. The mucosa shows hemorrhagic areas and congestion.

Intestines show congestion.

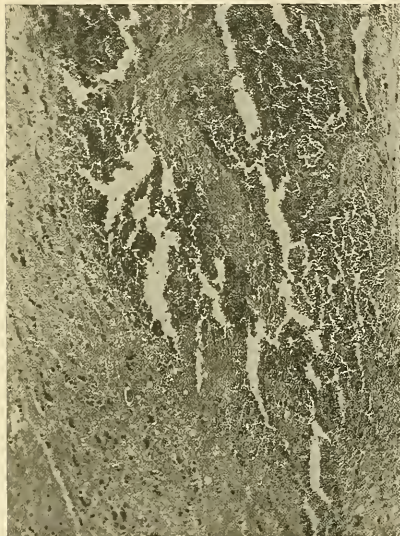


FIG. 7. LARGE INFILTRATING HEMORRHAGE IN PONS.



FIG. 8. PONS. HEMORRHAGE CONFINED TO PERIVASCULAR SPACE.

marked congestion of the blood vessels. The glandular tissue is of a glossy digested appearance.

Bladder.—Contains about 400 c.c. of straw colored urine. The bladder wall is trabeculated and at the trigone there are small hemorrhagic areas and the vessels are very prominent.

Vagina.—Contains a small amount of discharge. The cervix shows a reddened appearance and protruding from the external os there is a small reddish pedunculated polyp.

Uterus.—It is small and contracted. The cervical canal contains a small amount of mucus. The polyp is attached at the internal

MICROSCOPICAL EXAMINATION

Nerve Tissue. Cerebrum.—Sections of the gelatinous and hemorrhagic area described in the gross examination show a tumor growth which is a rather cellular glioma. The cells are small and have no alveolar or other orderly arrangement, except the typical roscate formation around some of the blood vessels. The blood vessels within the growth are very numerous. Most of them are thin-walled and congested while some of them show swelling and prolifera-

tion of the intimal endothelium. The margins of the tumor are not sharply outlined but merge indefinitely into the sur-

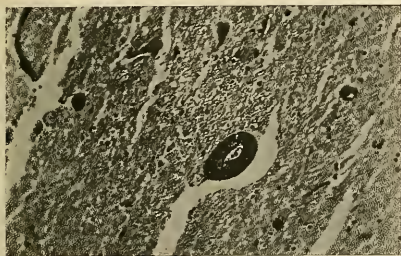


FIG. 9. SMALL HEMORRHAGES IN MEDULLA CONFINED TO THE VIRCHOW-ROBIN SPACE OF THE VESSEL WALL.

rounding brain tissue. In parts of the tumor mass there are necrotic areas which are the seat of former hemorrhages. There are also areas of hemorrhage which are of more recent origin and are in the process of organization. The largest ones, however, are of very recent occurrence, as the individual blood corpuscles are still preserved.

Brain Stem.—Throughout the pons and medulla there are numerous hemorrhages which vary in size from large ones which are plainly visible with the naked eye to small circumscribed hemorrhages which are only seen with the microscope. Most of the small hemorrhages are sharply outlined around the vessels and are confined to the perivascular space. In other instances they are limited by the adventitial covering and are simply collections of blood in the Virchow-Robin space which suggest a gradual oozing of blood through the damaged inner coats of the vessel wall. Sections of the cord were made and in the lumbar region small hemorrhages are found in the gray matter at the base of the anterior horn. The anterior horn cells throughout the cord are often swollen with eccentric nuclei, some showing central chromatolysis, but nowhere is satellitosis or neuronophagia observed. The cells of the pontine and medullary nuclei

show central chromatolysis and at times acute cloudy swelling, but the changes are not extreme and are not more marked than might be expected in any general infectious process. There is nowhere any lymphocytic infiltration except around some of the older hemorrhages in the substance of the tumor and here it is never marked.

Collections of polyblasts, amoeboid neuroglia cells, plasma cells or polymorphonuclear leucocytes are not found in any focal areas or around any of the vessels.

The lesions outside of the glioma are limited to simple hemorrhages and minor changes in the nerve cells of the brain stem and cord.

Heart.—The pericardium shows a thickening and hyalinization and an increase in fat. The

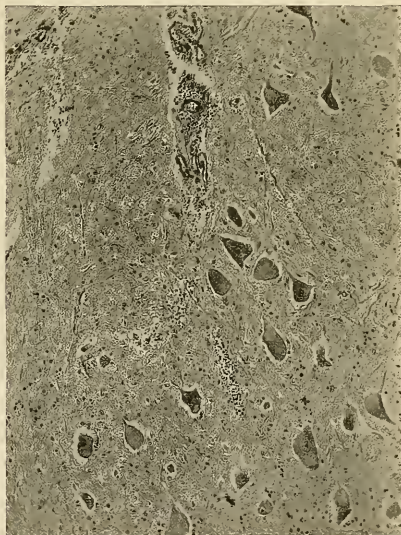


FIG. 10. HEMORRHAGES AND CHROMATOLYTIC CHANGES IN THE MOTOR CELLS OF THE CERVICAL REGION OF THE SPINAL CORD.

muscle cells stain poorly and irregularly and show a marked fragmentation. They are of a granular appearance. Some show marked

hypertrophy. The nuclei are irregular and stain poorly. Most are vesicular and where there is a marked fragmentation they are absent. There is a definite increase in connective tissue replacing quite extensive areas of heart muscle. This seems to be more marked around the blood vessels.

Lung.—The pleura shows nothing remarkable. The alveoli show a marked congestion with red blood cells which appear as pink hemorrhages. Some of the alveoli further are filled with a pink homogeneous-staining substance and some contain large numbers of lymphocytes. The alveolar walls are edematous and in many areas show a hyalinization and are of a necrotic appearance of the epithelium. The capillaries are distended with red blood cells and the larger blood vessels show a slight thickening.

Liver.—The capsule shows a slight thickening and hyalinization. The central veins are dilated and contain a granular debris and some red blood cells. There is a zone of hyalinized connective about these veins. The liver cells about the central veins are of a necrotic appearance and markedly fragmented. Very few of their nuclei stain. The cells about the periphery of the lobule stain more intensely but in their cytoplasm are large globules of probably fat giving the periphery of each lobule a vacuolized appearance. About the portal triad there is an increase of connective tissue with many lymphocytes infiltrating it. The sinusoids particularly about the central veins show a slight thickening.

Spleen.—The capsule and trabeculae are thickened. Few of the splenic corpuscles remain. There is a definite increase in red blood cells which occur in frank hemorrhages. Scattered throughout the sections, particularly in the hemorrhagic areas, is a greenish black pigment. Scattered throughout the spleen are groups of large mononuclear cells that appear

to be myeloblasts. Some of these contain several nuclei. The blood vessels of the center of the splenic corpuscles show a marked thickening, hyalinization and almost an obliteration. The larger blood vessels likewise show a marked thickening and hyalinization of the adventitia.

Pancreas.—The pancreatic acini show a decrease in size. The cytoplasm of the cells appears vacuolated. Only a few of the islands of Langerhans are evident. Many are apparently replaced by fibrous tissue. There is a marked increase in the interstitial tissue replacing many glands and giving the tissue a fibrous appearance. In areas this connective tissue appears necrotic. The blood vessels are sclerotic and many are obliterated.

Adrenal shows a thickening of the capsule and hyalinization. There is a congestion of the blood vessels in the medulla, otherwise there is nothing remarkable.

Kidney.—The capsule shows a thickening, and in areas it extends among the tubules. The glomeruli show a varying picture. Some are entirely replaced by fibrous tissue. Others show a thrombosis of the efferent vessel and a partial hyalinization of the tuft. Some tufts only appear swollen and show congestion. Bowman's capsule is thickened in almost every instance and many of the partially hyalinized tufts show adhesions to the capsule. The convoluted tubules also show a varying picture. The cells of some, although intact, are thinned and their nuclei appear vesicular and the cytoplasm stains poorly. In some there is a marked desquamation and in some a fragmentation of the cells. Here, few of the nuclei stain. The lumina of the tubules are filled with cellular debris and many are dilated and filled with a pink homogeneous-staining material. There is a marked increase in the interstitial tissue and here there is a dense, almost diffuse infiltration of lymphocytes. The large and small blood

Culture Taken at Autopsy from

- | | | |
|---------------------------------|---------------------------|-------------------|
| 1. Spinal fluid | { aërobic
anaërobic | } Culture sterile |
| 2. Inferior frontal gyrus | { aërobic
Pour plate | |
| 3. Uncinate gyrus | { anaërobic
Pour plate | |
| 4. Fluid from lateral ventricle | —Pour plate | |
| 5. Inferior temporal gyrus | —Blood agar plate | |
| 6. Pons | —Blood agar plate. | |

vessels show a thickening of all the layers; many are entirely obliterated.

Bladder.—There is an edema of the submucosa and muscularis. The muscularis appears slightly hypertrophied.

Ovary.—Shows a few semi-replaced corpora lutea. Otherwise the entire ovary has been replaced by fibrous tissue.

Uterus.—Shows nothing remarkable.

Breast.—Some glandular tissue remains but a considerable amount has been replaced by fibrous tissue in which there is a lymphocytic infiltration and numbers of the glands are cystic.

Thyroid.—Shows a slight increase in interstitial tissue in which areas appear which are hyalinized.

Aorta.—The intima shows a slight irregular thickening. In it are seen a few cholesterol clefts and a bluish homogeneous-staining material. This seems to extend quite diffusely through the media also. Some of the smooth muscle of the media has been replaced by fibrous tissue. The adventitia shows nothing remarkable.

Pituitary shows marked congestion.

Stomach.—Nothing remarkable.

Lymph Node shows edema and endothelial hyperplasia of lymph sinuses.

DISCUSSION

Hemorrhagic encephalitis has been frequently observed and specially described by Oppenheim, Strumpell, Lichtenstein and others. It is supposed to be the result of an infection and has been observed in a number of conditions, especially erysipelas, pneumonia, scarlet fever, measles, and frequently after influenza. Hassin reports a case following influenza and Dana and Schlapp observed one which was probably of malarial origin.

In this case all cultures were sterile and there was no evidence of any of the general infections with which hemorrhagic encephalitis is known to be associated. There was a history of influenza six months previously, but that would have no bearing on the present condition. The clinical history and neurological findings were indicative of an acute encephalitis which was

believed to be of the epidemic type; and this was the ante-mortem diagnosis.

The pathological findings in epidemic encephalitis usually show a moderate infiltration of the Virchow-Robin space of the blood vessels in the involved areas. This adventitial lymph space contains lymphocytes, polyblasts and some polymorphonuclear leucocytes. In the white, and to a less extent in the gray matter, there is an accumulation of these wandering cells and also a moderate reaction on the part of the ameboid neuroglia cells. There are also multiple hemorrhages and some alterations in the motor neurocytes.

In this case there was no marked reaction on the part of the wandering or the fixed tissue cells. There were, however, multiple hemorrhages, some of which were different from the usual type of hemorrhages, there being no break in the continuity of the vessel wall, the blood being confined to the Virchow-Robin space. Others were perivascular, that is, confined to the so-called space of His. The third type were of the usual infiltrative variety resulting from vascular ruptures.

To review the case in the light of the autopsy findings, we seem to have two distinct conditions present: first, a slumbering glioma which had given no symptoms until a hemorrhage occurred in its substance. This produced the initial convulsion and hemiplegia. The signs of pyramidal tract involvement rapidly cleared up but other symptoms supervened, namely, drowsiness, somnolence, headache and ptosis. After admission to the hospital the drowsiness soon became stuporous, the hemiplegia returned and death supervened in nine days. Pathologically the diagnosis is cerebral glioma and acute hemorrhagic encephalomyelitis. Occurring at the time when epidemic encephalitis is prevalent and in the absence of any general infectious disease, it seems possible that the multiple hemorrhages both in the tumor and in the brain and cord were produced by the condition termed epidemic encephalitis.

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GENERAL CONSIDERATION OF THE CRANIAL NERVES

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NEW YORK CITY

The peripheral nervous system in man has been divided into three separate categories, the cranial nerves, the spinal nerves and the vegetative system. The segregation of the vegetative system from the remainder of the nervous mechanism is easy to understand and defend—its anatomy is distinctly different from that of its parent stem. Within its own confines is set up a system which is definitely apart from the greater mechanism although it still demonstrates its connection with and its dependence upon the other system in the *rami communicantes* which pass between the two structures. The main system of the vegetative mechanism resides in the series of ganglia which lie ventral to the vertebral column, from which pass fibers forming peripheral plexuses and ganglia, and from which also subordinate fibers pass to their final destination. In its histology, the vegetative system shows its divergent character, the cells being in large part stellate, and of smaller size than the central nervous system neurones. The fibers are unmyelinated, or at best have but the slightest coating of myelin tissue. In its function it is widely different from the greater system, controlling the involuntary and vegetative processes of the body. The cells of the ganglionated cord and the paravertebral or peripheral ganglia are almost exclusively efferent in type. The fibers are both efferent and afferent, the cells of the afferent fibers not being located in the vegetative system ganglia but in the dorsal root ganglia with the ganglion cells of the somatic sensory

system. These differences readily set apart the vegetative from the voluntary nervous system.

The division, however, between the cranial and the spinal nerves is not based on nearly so satisfactory morphological and functional grounds. The criterion which is most patent is relatively inconsequential, that the cranial nerves are contained within the skull and emerge through various apertures and foramina in the base of the skull. Such a distinction is based wholly upon factors which in reality have nothing whatever to do with the essentials of the nervous system. The development of the bones of the skull is the result of mechanical and developmental factors quite apart from the real morphology of the central and peripheral nervous system. The development of a structure adapted to the offensive and defensive needs of the organism, to the acquisition, mastication and deglutition of food stuffs, and to the protection of organs called distance receptors, all have contributed to the formation of the skull. This bony framework in its development has grown about and encompassed certain of the nerves which we have found to be emerging from the neuraxis, and in so doing, the evolutionary process has marked off for the benefit of the future medical student certain nerves, which shall be henceforth nominated as cranial nerves, and shall be distinct from another series called the spinal nerves, in direct continuity with them but spared the baneful influence of the skull.

The differentiation between the two systems of peripheral nerves, however, is not quite so crude and superficial as the foregoing would seem to indicate, for the cranial nerves do differ in their structure and function from the spinal nerves. This difference is one of degree, whereas the distinction between the voluntary and involuntary system is one much more fundamental.

Various classifications have been devised for the cranial nerves; the first intelligible one was that of Willis, whose name has been so long attached to the anastomotic circle from which the blood supply is provided for the brain. His classification is as follows:

1. Olfactory
2. Optic
3. Oculomotorius (communis)
4. Trochlearis (patheticus)
5. Trigeminus
6. Abducens
7. Facio-acousticus
8. Glossopharyngeal-vago-spinal
9. Hypoglossal
10. Suboccipital (1st spinal).

This classification was later modified by Sommering, as follows, and is the one which is now in common use:

1. Olfactory
2. Optic
3. Oculomotor
4. Trochlear
5. Trigeminus or trifacial
6. Abducens
7. Facialis
8. Auditory
9. Glossopharyngeal
10. Vagus
11. Spinal accessory
12. Hypoglossal.

On the basis of the components of the cranial nerves another classification has been formulated:

- I. Nerves of special sense
 1. Olfactory
 2. Optic

3. Auditory

II. Sensory—motor or mixed nerves

1. Trigeminus
2. Facial
3. Glossopharyngeal
4. Vagus

III. Motor nerves

1. Oculomotor
2. Trochlear
3. Abducens
4. Spinal accessory
5. Hypoglossal

Before beginning the consideration of the individual nerves and the origin of the musculature which they will be found to innervate, it may be well for us to analyse the individual constituent parts of a typical spinal nerve in order that we may have a relatively simple pattern with which to compare the more complicated cranial nerves.

THE COMPONENT THEORY

The components of a spinal nerve may be divided under four heads:

1. Somatic motor
2. Somatic sensory
3. Splanchnic motor
4. Splanchnic sensory.

This is called the component theory of the nervous system.

The somatic motor system comprises those nerve cells whose axonal processes supply muscles which have developed from the body segment or myotome. They are located in the ventral horn of the gray matter and are large multipolar cells, the typical cells of the ventral horns. Their fibers leave the gray matter and sweeping ventral and lateral emerge from the ventrolateral aspect of the spinal cord as the ventral roots.

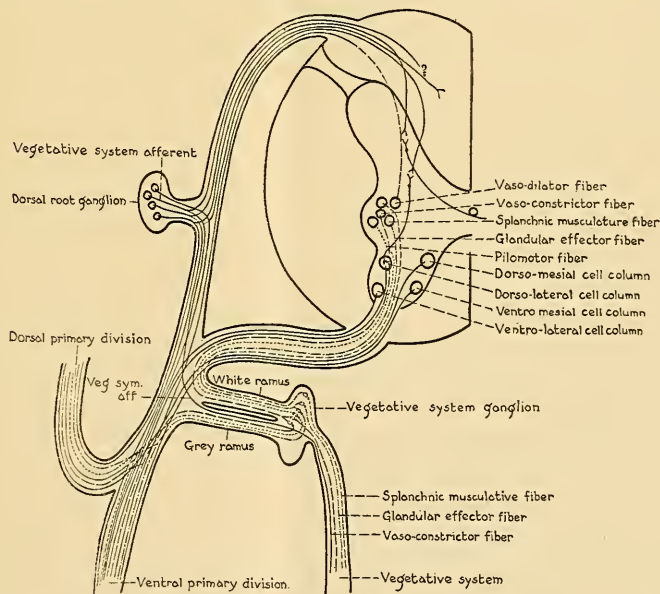
The somatic sensory system comprises the sensory mechanism of the body whose peripheral processes end in the skin, the mucocutaneous junctions and the deeper mesenchymal structures, such as muscles, bones, joints, etc. Their cells are unipolar cells which are aggregated to form the dorsal root ganglia lying on the dorsal roots.

The central processes of these cells form the dorsal roots, and entering the cord end as fibers which pass up and down, across the cord, or at once form synapses with dorsal horn cells. From these cells secondary axones pass either up or down or across the cord to end in synapses with either tertiary sensory neurones, motor or associational neurones.

The splanchnic motor system comprises a group of motor cells smaller and less richly supplied with processes. These cells lie in the lateral horn of the gray matter, and their peripheral processes pass out through the ventral roots with the somatic motor

or the secondary neurones may turn back as the gray *rami communicantes*, fuse with the peripheral nerves and pass as vasomotor glandular effector and pilomotor nerves to the periphery.

The splanchnic sensory system is composed of fibers which arise in the organs of the thoracic and abdominal organs, pass through the plexuses of the vegetative system over the gray *rami communicantes* and thence to the dorsal roots. Their cells lie in the dorsal root ganglia and their central processes enter the spinal cord and pass thence over little known pathways through the cord either upward, downward or



THE COMPONENT CONSTITUTION OF THE NERVOUS SYSTEM.

fibers. They do not continue with these fibers, but shortly after leaving the cord, split off as the white *rami communicantes* and pass to the ganglionated cord of the vegetative system. There they may terminate around cells which in turn send their axones outward into the vegetative system,

across. That some of them eventually reach the cortex can not be denied, for we have certain definite if intangible visceral sensations connected with the gastrointestinal and genito-urinary tracts.

These components form certain definite connections within the spinal cord.

In relation with the cells giving origin to the somatic motor fibers, there end axones which descend from above, from the cortex, called the pyramidal system, from the infracortical centers, the basal ganglia, the cerebellum, etc., called the extrapyramidal system, and from adjacent ipsilateral and contralateral segments.

The somatic sensory fibers upon entering the cord may pass upwards, downwards or across the cord and end in synapses with either intercalated cells in their pathway toward the cortex or with motor cells in order to produce reflex action.

The splanchnic motor system in all probability receives some sort of control from above, but its exact nature and character are not known.

The splanchnic sensory system is largely subconscious in nature and the anatomy of their connections and course is not known.

MYOTOMIC AND BRANCHIOMERIC SEGMENTATION

The process which has complicated the problem in the head region is the development in that position of two very definite sets of musculature, one in connection with the externalizing mechanism, the great somatic musculature represented in the body by the trunk and limb muscles and in the head by the eye and tongue muscles, the somatic musculature, and a secondary set of muscles called the branchiomic muscles. These muscles in the lower animals are represented by the musculature of the gills, the muscles of the masticatory apparatus and the upper part of the alimentary canal. These are involuntary in character and are a part of the unstriped musculature of the organism. In the higher forms, however, in the interests of greater efficiency, and with modified functions, these muscles have lost their involuntary, unstriped characteristics and have become voluntary striated muscles. Such a superficial change phylogenetically speaking does not scratch the surface of the much less impressionable nervous system which retains its older, more primitive arrangement; and thus we find the peripheral

structures assuming an altered form and function, whereas the central connections tend to retain their older form. This double segmentation in the head region gives us a basic segmental myotomic series with a superadded and complicating branchiomic segmentation. This adaptation, however, of muscles is not complete and we find the voluntary branchiomic merging into the involuntary splanchnic musculature in the muscles connecting the head and the trunk, the sterno-cleido-mastoid and the trapezius.

In the development of the embryo there appear on the lateral surface of the neck region a number of elevations and depressions called branchial arches and clefts, which represent the gill clefts of the fish. In these arches there develops from the mesoderm a condensation of tissue which will form bone and muscle tissue. The arches are four in number and from the bony formation within them the two upper arches are given other names; the first branchial arch containing the developing mandible is also called the mandibular arch, and the second arch containing the hyoid complex, the hyoid arch. Each one of these arches will develop a certain set of muscles and with each arch will be found a cranial nerve. The arches occur between the positions of the body segments and thus are intersegmental, and the vessels and nerves which enter them lose to a certain extent their segmental character and become intersegmental, thus not being comparable to the segmental nerves and vessels of the trunk.

THE MYOTOMES

There are supposed to be nine myotomes or body segments in the head region, some of them persisting into the adult form, some fusing and others completely disappearing. The first three, counting from the cephalic extremity caudad, form the muscles of the orbit, those which move the eyeball, the four recti and the two obliques. The first myotome forms the superior, inferior and internal rectus and the inferior oblique, and has as its nerve the third cranial nerve or the

nervus oculomotorius; the second myotome forms the superior oblique and has as its nerve the fourth cranial nerve or trochlearis, while the third myotome forms the external oblique and has as its nerve the sixth cranial nerve or the abducens. The next three myotomes, IV to VI inclusive, have disappeared and the ventral roots originally in connection with them have disappeared; the last three VII to IX inclusive, have fused to form the voluntary musculature of the tongue. By many these last three dermatomes are considered to be spinal in origin and have only later been incorporated in the cranial series. These fused myotomes have the twelfth cranial nerve or the hypoglossal nerve, which represents four or five nerve trunks fused into a single bundle. The cervical character of this nerve is further evidenced by a bundle of fibers joining the nerve from the first and second spinal nerves, which later splits off as the descendens hypoglossi and joins with the descendens cervicalis to form the ansa hypoglossi, and supplies myotomic trunk muscles in the neck region, the sternohyoid and omo-hyoid.

THE BRANCHIOMERES

As we found the segmental nerves of the neuraxis, III, IV and VI, to be in definite relationship with the myotomic segment series, so we find the intersegmental or splanchnic series in relationship with the several branchial clefts and structures derived from these clefts.

The fifth cranial nerve in its motor portion is the essential nerve of the mandibular arch, its sensory portion combining, as we shall see, all of the general sensory area of the head regions, both cutaneous and mucosal. The muscles of the mandibular arch and the migrated mylo-hyoid and anterior belly of the digastric muscle constitute the muscles of mastication, and they arise from the splanchnic mesoblast within the mandibular arch.

The seventh cranial nerve is the nerve of the second branchial or the hyoid arch and

from its musculature develop the stapedius, the stylo-hyoid and posterior belly of the digastric, the epicranial, facial and platysma myoides group—all muscles derived from the sphincter colli group of muscles, but which in the course of development have become the muscles which pre-eminently supply the externalizing mechanism for our emotions, and the play of features which forms such an intimate part of our social contact with our fellows.

This musculature derives its nerve supply from the facial fibers which arise in the facial nucleus and correspond with the lateral series of cranial motor nerve nuclei. They supply branchiomic muscles. In addition to this efferent outflow there are glandular effector fibers which emerge from the nerve as a part of the great superficial petrosal nerve passing to the sphenopalatine ganglion and tympanic fibers which eventually reach the submaxillary ganglion. The sensory portion of the nerve representing the taste-bud supply of the anterior two-thirds of the tongue falls under the category of splanchnic afferent and ends about cells which we will find form the uppermost part of the great splanchnic afferent receiving station in the medulla, the fasciculus solitarius.

The ninth cranial nerve, or glossopharyngeus, is considered to be the nerve of the third branchial arch or the so-called thyro-hyoid arch. Its muscular portion supplies the stylo-pharyngeus and the superior constrictor of the pharynx. In addition to supplying the superior constrictor of the pharynx the glossopharyngeus nerve supplies fibers which join with fibers from the vegetative system to form the pharyngeal plexus, which as one of its functions supplies the involuntary unstriped middle and lower constrictors of the pharynx. The glossopharyngeus thus acts as a functional and morphological bridge connecting the voluntary and the involuntary nervous systems. Its sensory portion, mainly taste for the posterior third of the tongue, is splanchnic afferent and the fibers find their end station

in that part of the fasciculus solitarius lying caudad to the portion belonging to the seventh cranial nerve.

The tenth cranial nerve, or vagus, represents the fusion of all the branchial nerves caudad to the ninth, and it carries over into the involuntary category the process initiated by the glossopharyngeus supplying the post-oral intestinal tract as far as the large intestine. In its afferent system are combined two constituents, the somatic, represented by the small auricular branch, and the splanchnic represented by the enormous distribution of terminal filaments to the viscera of the thoracic and abdominal cavities. The superior laryngeal nerve is the nerve of the fourth branchial arch, and the inferior laryngeal, a branch of the vagus, supplies the subordinate fifth branchial arch.

The somatic sensory portion of the vagus distribution probably joins the spinal fifth within the neuraxis and ends in relation with cells of the substantia gelatinosa. The splanchnic afferent vagal component appropriates the remaining portion of the fasciculus solitarius caudad to the glossopharyngeal portion of that column and the gray matter associated with it.

The secondary pathways of these fibers are but little known, but it is most probable that the somatic afferents are all eventually assembled together with those rising up from the spinal cord and enter the internal capsule in one bundle. The termination of the secondary splanchnic afferents is not known.

The eleventh cranial nerve is composed of two portions, the accessory, which is really a supplementary part of the vagus system, and a spinal portion which supplies the sternocleido-mastoid and the trapezius muscles. In addition to this innervation, these muscles are also supplied directly by ventral root fibers from the upper cervical nerves, so that these muscles receive two forms of nerve supply, branchiomic from the spinal accessory and myotomic from the upper cervical roots. The muscles themselves joining the head to the trunk represent the

transition from branchiomic to myotomic musculature.

THE CONSTITUTION OF THE GRAY MATTER

The relative constitution of the gray matter is of considerable importance in homologizing the spinal with the cranial series. The cell groups which give origin to the somatic motor components comprise the ventral horn cells, the mesial motor cell column. This column of cells, as its name indicates, is located ventrally and mesially, and as we pass upward we will find that the mesial motor cell column, although it becomes discontinuous, is represented in the groups of cells which give origin to the hypoglossal, the abducens, the trochlearis and the oculomotorius nerves, thus providing additional proof of the somatic character of these nerves. In like fashion the cell groups which give origin to the splanchnic efferent or motor fibers are located in the central and lateral portions of the gray matter, and if they be followed cephalad they will be found to be continuous with an interrupted series of motor nuclei called the lateral cell column, giving origin to the motor roots of the eleventh, tenth, ninth, seventh and fifth cranial nerves. This cell column is the splanchnic motor cell column and is further evidence as to the character of the motor innervation of these nerves.

The general somatic sensory end station of the cranial nerves in the intra-axial gray matter corresponds with the dorsal horns of the spinal gray matter. The substantia gelatinosa of Rolando, the descending root of the fifth nerve beginning above at the upper level of the pons, ends as a cap over the uppermost portion of the dorsal horn gray matter of the cord in the second cervical segment.

The acoustic mechanism is probably a modification of the general somatic afferent system, and ends in the tuberculum acusticum which is homologous with a part of the dorsal horn of the spinal cord.

The sensory end stations of the splanchnic components of the seventh, ninth and tenth cranial nerves form a series of nuclei which

are discontinuous but correspond in general to a prolongation upward of the gray matter of the posterior horn and that portion of the body of the gray matter which receives the posterior horn. Its greatest aggregation forms the nucleus of the fasciculus solitarius which receives in turn from above downward the splanchnic afferent fibers of the seventh, ninth and tenth cranial nerves. The tenth in addition to the nucleus of the fasciculus solitarius possesses a dorsal sensory nucleus which lies in the gray matter in the floor of the fourth ventricle.

The rearrangement of the white fibers in the medulla is probably largely responsible for the breaking up of the gray substance as it is found in the spinal cord. This disturbance is contributed to, also by the opening and closing of the fourth ventricle. The gap in the ventral motor cell column between the nuclei of the sixth and twelfth nerve nuclei is due to the disappearance in that region of the somatic myotomes which would have received their nerve fibers from that region. The gap between the fourth and sixth is

explained by the migration downward of the sixth nerve nucleus which is situated more cephalically during embryological development.

The lateral cell column, although broken up by interlacing bundles of fibers, presents a fairly continuous line of gray matter from the mesencephalic root of the fifth nerve all the way down to the lower limit of the decussation of the fillet where the nucleus ambiguus becomes broken up by the beginning decussation of the pyramidal tract.

THE CRANIAL NERVE GANGLIA

The ganglia in relationship with the cranial nerves are of two types, stationary and vagrant. The stationary are found only on afferent nerves and are strictly homologous with the dorsal root ganglia found on the dorsal roots of the spinal nerves. These stationary ganglia include the Gasserian ganglion of the fifth nerve, the geniculate ganglion of the seventh nerve, the ganglia of Scarpa and Corti of the eighth nerve, the superior and petrosal ganglia of

SUMMARY OF THE COMPONENTS OF THE CRANIAL NERVES

<i>Nerve</i>	<i>Somatic Efferent</i>	<i>Somatic Afferent</i>	<i>Splanchnic Efferent</i>	<i>Splanchnic Afferent</i>
Third	To orbital muscles	—	Ciliary and Sphincter Iridis	—
Fourth	To sup. obl.	—	—	—
Fifth	—	Sensory root	Motor root	Sensory root
Sixth	To ext. obl.	—	—	—
Seventh	—	—	Motor root facial petrosal. Tympanic fibers to submaxillary ganglion.	Sensory root
Eighth	—	Auditory	—	—
Ninth	—	—	Stylo-Pharyngeus Pharyngeal muscles. Small superficial petrosal to otic ganglion.	Sensory root
Tenth	—	Auricular	Laryngeal muscles. Muscles of intestinal canal.	From viscera
Eleventh	Sterno-cleido-mastoid and trapezius	—	Sterno - cleido - mastoid and trapezius. Vagal accessory.	—
Twelfth	Tongue	—	—	—

the ninth nerve, the jugular ganglion and possibly the ganglion nodosum of the tenth nerve. All these ganglia present unipolar cells, with the exception of the acoustic ganglion which has bipolar cells. The hypoglossal nerve presents early in its history a ganglion, the ganglion of Froriep, which indicates an early typical segmental character in the twelfth nerve. This ganglion disappears, and its cells and fibers are supposed to become incorporated with the Gasserian ganglion and the fifth nerve—as is also the case with the dorsal root and ganglion of the first cervical nerve. Occasional groups of similar ganglionic crest cells have been found along the course of the third, fourth and sixth nerves, indicating their early typical segmental characteristics.

The vagrant ganglia are homologous with the prevertebral ganglia of the vegetative system type. The vagrant ganglia are the ciliary in connection with the third nerve, the sphenopalatine, otic and submaxillary in connection with the fifth nerve. They receive roots from various other sources, the vegetative system, and other cranial nerves, and probably receive contribution from a number of cerebral segments.

THE NERVES OF SPECIAL SENSE

We can now undertake a consideration of the nerves which have so far been neglected in our review of the cranial nerves, namely, the olfactory, optic and auditory nerves. These are nerves of special sense and they are in connection with organs whose special function it is to transform stimuli coming from a distance into nerve impulses which will convey certain specialized messages to the brain. This introduces the subject of encephalization and the development of distance receptors, one of the most fundamental considerations in our phylogenetic history which in brief resulted from the necessity of determining one end of the body as the head end which would progress in advance of the tail end. Secondary to this determination there began to be developed

organs which placed the organism in contact with its environment at distances further and further removed from actual contact, first through vibrissae or antennae, then through specialized photo-sensitive cells and finally sight-perceiving organs. In like manner organs developed which were capable of receiving stimuli of smell and hearing. These three sense systems have developed largely in a similar way, by an evagination from the central neuraxis coming into contact with an invagination from the ectoderm, the two fusing and forming the complicated special sense organs. In the most anterior portion of the body, the evagination forms the olfactory lobe, tract and bulb, the cutaneous component being the olfactory placode, and in the fusion of the two is developed the olfactory membrane and the mitral cells of the olfactory bulb. Similarly the optic evagination forming the optic stalk and cup, establishing contact with the ectoderm, produces the differentiation of the lens and other mesodermal constituents of the optic mechanism. In the acoustic apparatus we have the neuraxial evagination, establishing contact with the ectodermal otocyst with the resulting formation of auditory and vestibular mechanisms. The formation of the last is not strictly homologous with the first two, for in the last we have a definite outgrowth from the neural crest to form the two ganglia, the ganglia of Scarpa on the vestibular root and the ganglion of Corti on the acoustic root. The olfactory and optic apparatus is not homologous in any way with the typical spinal nerve, for in both instances we have a primitively hollow evagination of the brain wall itself, establishing secondary and tertiary connections with the ectodermal placodal formation, so that the olfactory and the optic tracts represent secondary and tertiary pathways. This view is further substantiated by the fact that like the intra-axial pathways, the fibers of the olfactory and optic tracts possess no sheaths of Schwann.

THE CRANIAL NERVES AS SIGNPOSTS
IN DIAGNOSIS

The cranial nerves are of extreme importance in diagnosis in that they are signposts pointing to the location of lesions involving the neuraxis. The tracts of the spinal cord are long, and an interruption at any point along their course will give approximately the same phenomena. The character of the neuraxis with its closely packed constituents can scarcely suffer in one or two components only, and a lesion of any magnitude will almost of a certainty produce interferences with one or more systems of fibers or cell groups. In the spinal

cord we have a great number of crossroads at which lesions may produce disturbances in the long ascending and descending tracts, and at the same time interfere with certain of the spinal nerves, throwing out of function definite cell groups controlling certain muscles and centers. So in the brain stem, lesions will involve the long tracts and the cranial nerves, and the localization of a lesion will depend in large part upon the identification of the exact crossroad at which the lesion has taken place. To do this a knowledge of the anatomy of the course of the tracts and the intra-axial pathways of the cranial nerves will serve to supply the data whereon the diagnosis may be based.

REPORT OF A CASE OF EPIDEMIC POLIOENCEPHALITIS WITH TIC-LIKE MOVEMENTS

BY GEORGE C. ANDREWS, M.D.

NEW YORK CITY

Patient, male, aged thirty-nine, had always been well with the exception of dysentery twelve years ago and pneumonia six years ago. He denied venereal infection. He came of healthy family stock, there being no chronic diseases or nervous or mental diseases in the family.

On the 1st of last February he was suddenly stricken with fever, malaise, cough and sore throat. His fever was high. On the fourth day of his illness he was taken to the St. Johns Hospital, Yonkers, and there told that he had influenza. For forty-eight hours after he entered the hospital he was irrational, had high fever, and threw himself about in bed, calling out. Once during this time he had to be catheterized. Then he became stuporous and lethargic. His vision became blurred and he had diplopia. He slept much of the time, and had to be awakened for his meals. After four weeks he improved enough to go home.

On leaving the hospital he felt generally weak throughout his body, but not any weaker on one side than on the other. He lay about at home for a few days and again became somnolent and lethargic. He "slept all night and all day," and had to be awakened for each meal. He remained profoundly lethargic for about two weeks, after which he gradually improved. The blurring of vision and diplopia disappeared.

At no time did he have any difficulty in swallowing, or incontinence. He had no headaches, nausea or vomiting. His neck was not stiff.

In May, four months after the initial illness, he was enough improved to go back to work. He still felt rather weak, and his employers therefore gave him a job at a machine called a shaper. This machine had a bar which moved to and fro on his right.

Sometimes he would be watching this bar and would momentarily lose his balance and tend to fall to the right. He was still quite sleepy and was generally weak. The weather became bad, and he felt worse and weaker, and the right side of his face began to twitch. The twitching occurred when the handle on his right moved toward him. At first it was infrequent, but later it became very constant. At times on arising from his seat at the machine he felt weak on the right side of his body and as if he were about to fall to the right.

He came to the Vanderbilt Clinic six months after his initial illness, complaining of trouble with his speech, with twitching of the right side of his face and turning of his head to the right and weakness of the right side of his body. The first symptom he had noticed was weakness in the right side of his neck. When he sat down he would hold his head in his hands because of the tendency of his head to fall to the right.

General physical examination was negative except for a slightly enlarged heart and widely spaced teeth.

Neurological examination revealed as follows: The head was usually held a little rotated to the right and the patient tended to look to the right. The head was also held slightly forward and the chin was drawn in. The right shoulder was lower than the left, and there was some scoliosis, with convexity to the left. The gait showed a tendency to lean to the right after continued walking. He had always been right-handed. His equilibratory and non-equibratory coordination were good. His speech showed some hesitation in saying "Methodist Episcopal," but the other test phrases were well rendered. There was a spasmodic twitching of the right sterno-cleido-mastoid, deltoid

and platysma muscles, and there was an element of control and suggestibility in these movements. By placing his head against the wall he could stop the twitchings. A regular fine rapid tremor existed in both extended hands. This was increased by attention and was non-motorfacient. His deep and superficial reflexes were all present and equal. There were no pathological reflexes. The muscle strength was normal except that it was more effort for him to hold his head rotated to the left than to hold it rotated to the right. The muscle status was good. There were no abnormal associated movements. The general sensory examination was normal throughout.

The cranial nerves were as follows: Vision was 20/20. When first examined there was some blurring of the nasal side of the left disc and the veins of the retina were slightly tortuous. The left disc was suspicious of a mild optic neuritis. Both pupils were a little sluggish, especially the right. There was slight exophthalmos. In voluntary movements of the eyes there was slight weakness of the right internal rectus muscle, and there was a Moebius, the right eye showing insufficient convergence. There were slow irregular nystagmoid movements on looking laterally to the right or to the left. The right

palpebral fissure was a little larger than the left. Whether there was slight ptosis of the left upper lid was difficult to determine. The other cranial nerves were normal.

Both blood and spinal fluid Wassermanns were negative. The spinal fluid contained 6 cells per c.m. There was no globulin reaction; the colloidal gold was oooooooooo.

On subsequent examination the patient's condition was unchanged except that the tic-like movements had spread to his right arm and leg, and slightly to his left arm. The suspicious appearance of his left optic disc had disappeared. The same element of control existed in the movements. By concentration he could stop them for a time and by bracing himself against the wall he could stop them indefinitely.

The case seemed unquestionably to have been one of encephalitis lethargica. The tic-like movements were the development of a defensive movement originating from the machine at which he worked. They were purely functional, and would probably never have occurred had his convalescence been complete when he returned to his work. The convalescence of cases of lethargic encephalitis is tedious and protracted. For a long time after the acute illness they are subject to intercurrent disturbances unless properly cared for.

MARIE'S HEREDO-CEREBELLAR ATAXIA

DISCUSSION AND REPORT OF A CASE*

BY MICHAEL OSNATO, M.D.

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¹ Marie in 1893 reported a group of cases described by Nonne, Fraser and others, adding some cases of his own. He attempted to build up the clinical entity, which he called heredo-cerebellar ataxia, on a definite pathological basis. The pathological substratum for this condition was said to be a hereditary atrophy of the cerebellum. Marie attempted definitely to separate the symptomatology and pathology of this condition from Friedreich's ataxia and other atrophies and degenerations of the cerebellum and its pathways. Since that time numerous cases have been reported of cerebellar ataxia with a symptom complex exactly like the case described by Marie which were not either clinically or pathologically absolutely identical. For instance, cases have been described which were not familial, and others upon autopsy ² have shown symmetrical atrophy of the cerebellum including its peduncles, olives, etc., with a clinical appearance similar to the case described by Marie, but occurring in individual members of a family and without heredity. In fact, Dejerine ³ states that this disease is a systemic primary atrophy of the cerebellum which may be produced by many processes of various kinds either in the cerebellum or its tracts. Both Oppenheim and Dejerine feel that no sharp dividing line "can be drawn between the two forms of ataxia, the spinal (Friedreich's) or the cerebellar (Marie's)." Both types have been observed in the various members of one

family.⁴ This view is also taken by Jelliffe.⁵ Not only does it appear impossible to look on these cases as definite clinical entities of a degenerative or atrophic pathological nature occurring in families or determined by heredity, but there are a number of cases reported as progressive atrophy of the cerebellum which undoubtedly belong to this same group. The most recent one of this type reported with autopsy is that of Archambault.⁶ This case resembles in a great many features the case described below, but nevertheless our patient seems to belong to the group described by Marie in that there is a definite familial occurrence. Marie's cases, however, began either in early childhood or about the age of twenty. Archambault's case developed after the age of forty. Our patient also began to have his most prominent symptoms about the age of forty. The anatomical lesions in Archambault's case were confined to the cerebellar cortex with only insignificant changes in the secondary nuclei of the cerebellum, the olives, the spino-cerebellar tracts and the columns of Goll. The lesions were most marked in the cerebellar folia. All three layers of the cortex were involved, but in many parts of the cerebellum only the layer of Purkinje cells was affected. This last finding, namely, disappearance or degeneration of the Purkinje cells was the most constant feature. The same finding occurred in the cases published by Thomas, Dejerine, Murri and Rossi, quoted by the author. In Archambault's case the blood vessels of the

¹*Semaine Méd.* 1893.

²Dejerine and Thomas (quoted by Oppenheim, p. 1051), "Text Book of Nervous Diseases," English translation.

³Dejerine, "Séminologie des affections du système nerveux," p. 426.

⁴Nonni and Mingazzini (quoted by Oppenheim, p. 1051).

⁵Jelliffe and White, "Text Book of Nervous Diseases," 1915, p. 415.

⁶*Tr. Am. Neurol. Assn.*, 1918, p. 79.

* This patient was presented at a clinical conference before the staff of the Neurological Department of Columbia University on Oct. 15, 1917, and on Dec. 11, 1919.

leptomeninges showed markedly thickened walls, and particularly in the molecular zone cellular infiltration was found. The author feels that while the other writers whom he quotes ascribe their cases entirely to a primary parenchymatous atrophy, in his case at least there was sufficient evidence for his belief that the condition was secondary to vascular disease. As this author states: "It is therefore believed that in the genesis of cortical parenchymatous atrophy of the cerebellum, sclerosis of the meningeal vessels plays an important part." There are of course other views concerning the pathogenesis of these cerebellar atrophies.⁷ Tanzi and Lugaro state that until now forced efforts have been made to classify these cases according to the anatomical site of the lesion, and in this way create a variety of clinical types, usually carrying the author's name. However, the importance of these cases is that they serve as types or models about which other more or less similar cases may be grouped; but it is impossible to do this completely. In fact, the typical cases are the exception. Many aberrant types of varied character serve as transition types connecting these various groups together. They believe that Friedreich's ataxia and Marie's heredo-cerebellar ataxia cannot be either clinically or pathologically separated absolutely. Neither one of these problems can be entirely separated from that of hereditary syphilis, which acts as the cause of a simple hereditary degeneration, or as an active syphilitic process attacking the central nervous system and giving rise to the clinical syndrome. These authors hold the view that all familial and hereditary degenerative diseases have an accidental factor so far as their location is concerned. Certainly this would appear to be the case with Friedreich's ataxia and Marie's heredo-cerebellar ataxia. For instance, Oppenheim⁸ states that the pathological anatomy of Friedreich's ataxia "in spite of a great num-

ber of postmortem results and exhaustive examinations" has caused wide differences of opinion. In Friedreich's ataxia there is usually degeneration of the columns of Goll and Burdack—both lateral and cerebellar tracts, of Clarke's columns, the pyramidal tracts, and sometimes Gowers' tract. On the other hand, Oppenheim quotes Senator, Menzel and Archambault as finding a congenital atrophy of the cerebellum and an inconstant and uncertain condition in the spinal cord. Others have found in addition to the atrophy of the cerebellum a degeneration of the tracts mentioned above in the spinal cord, with also an ependymitis in the floor of the fourth ventricle. Oppenheim (page 198) agrees that "the distinction between Friedreich's disease and Marie's cerebellar ataxia cannot be definitely drawn from the symptomatology any more than the anatomical basis." This appears to be the conclusion of Bing.⁹ The views of Mingazzini¹⁰ are interesting. He groups these two conditions with amyotrophic lateral sclerosis and with the congenital palsies of the optic, facial and oculo-motor nerves. To these last he ascribes an aphasia of their respective nuclei of origin as the causative factor. He states: "No disease lends itself to a demonstration of the principles of congenital deficiency of nervous elements so readily as infantile general paresis, in which the persistence within the superficial layers of the cortex, of embryonal or fetal pyramidal cells has been found." Cajal has also shown the persistence of these embryonal cells in true epilepsy. The deficiency of function in these cases is a direct expression of the disturbance in the evolution of the cells. These embryonal and fetal pyramidal cells have also been found in infantile palsies and in the spinal cord of paralyzed adults. Mingazzini believes that abnormalities of size and shape and distribution of neurons and fiber tracts predisposes to degeneration and other pathological processes.

⁷ Oppenheim, "Treatise of Mental Diseases," 1916, Vol. 2, p. 300.

⁸ Ibid., p. 196.

⁹ Bing, "Text Book of Nervous Diseases," 1915, Eng. Translation, p. 129.

¹⁰ Mingazzini, "Anatomia Clinica dei Centri Nervosi," p. 911.

REPORT OF A CASE

HISTORY.—The patient J. H. C. was seen on September 9, 1917, when he was forty-eight years of age. He is married; was born in this country. Most of the information concerning the case was obtained from his wife, father and sister. We were able to see and speak with two brothers and a sister. Only the patient and the sister could be thoroughly examined.

Complaints.—The patient complains of failing vision, disturbance of speech and a staggering drunken gait. The first symptom was noticed about five or six years prior to the first examination. The onset had been rather gradual, but the character of the first symptom had to do almost entirely with awkwardness and unsteadiness of gait. The disease has gradually progressed without remissions.

Personal History.—All venereal diseases are denied except an attack of gonorrhea twenty-eight years ago. The patient never smoked or drank and never took any drugs. He never worked with lead or mercury, and was not interested in painting. He has never had any serious illness. He has been married for twenty-five years, and the wife has never been pregnant. The patient developed normally, following a normal pregnancy and labor. He walked and had his first teeth about the age of ten months. He walked very well. The father does not remember that the child was particularly awkward nor did the patient stumble and fall during infancy any more frequently than other children. The father does insist however that as a boy he was never skillful mechanically, and that while he was active he never attained even an ordinary success in the usual boys' games. He was never a good student, and the father believes that he has not been average in his mentality all his life and was, perhaps, below par. The patient's wife says that her husband has not been bright or even ordinarily intelligent. She insists that he was always rather slow-witted and dull. She recalls that he has never been mechanically inclined, has always been

awkward and has never learned to run or dance or play base ball or foot ball. She has known him since he was twenty years old. He was never accomplished in doing things that required skill. She remembers that he always took a little longer to dress and undress than she thought he should. He is a laundryman by trade and in working with him in their business she noticed that he was never as skilled in sorting and marking laundry as their employees, although he had worked in the same business all his life. No ataxia, dysmetria or asynergia and no speech or gait disturbances were ever noticed until he was about forty years of age. It is important to note the father's statement that between the ages of ten and sixteen this boy had six separate fractures occurring in the bones of both forearms. This is certainly an unusual thing, especially when one finds that although the boy was rather awkward and fell and stumbled frequently during these years, yet he never fractured his legs. The father states that prior to the age of ten the child did not fall nor was he exceptionally unsteady on his feet; but beginning soon after that age he was so awkward at play and when running that he stumbled and fell frequently. Concerning these fractures the author begs to call attention to some findings in the bones of the lower extremities reported in "An analysis of seven cases of Friedreich's ataxia"¹¹, in some of which *x-ray* examinations showed rarefying changes in the bones of both lower extremities and in many of the small bones of both feet.

Many inquiries were made concerning the possibility that disturbance with the patient's co-ordinating mechanisms might have been visible a good deal earlier than the date of onset as given by the patient and his relatives. The patient's father however insists that the unsteadiness of gait, the general awkwardness and the patient's present symptoms did not begin before he reached the age of forty. Nevertheless, the fact that this boy stumbled and fell while

¹¹*N. York M. J.*, Feb. 2 and 9, 1918.

running so often during his boyhood, is significant. There is no history that he ever staggered or was unsteady in walking; yet it is probably true that the various games played by children requiring closing of the eyes or considerable running made an unusual demand for purely equilibratory proprioceptive mechanisms upon him, bringing out the latent weakness in the organ presiding over this function, the same weakness at that time not being sufficiently great to produce symptoms when the boy was at rest or walking.

The wife first noticed that the patient was getting laughably awkward when he climbed in and out of bed, and that he fumbled and was uncertain in the movements of his hands and fingers in dressing and undressing. He was worse when he tried to do these things in the dark, but was not a great deal better in broad daylight. The patient says that he felt at this time that while walking his feet were being dug into the ground or that they were glued to the ground. He was very fond of swimming, and the wife says that he was a good swimmer and that when he was in the water all traces of unsteadiness disappeared; and until he became almost bedridden he was able to swim nearly as well as he did before he became ill. The speech defect began about one year and a half before the date of the first examination. His relatives noticed that he spoke in a slow, monotonous, expressionless, tired sort of way with a tendency to indistinct pronunciation. The voice became high pitched. Gradually the unsteadiness and staggering gait became so pronounced that he could not walk without assistance. At the same time as the speech defect showed itself, he noticed that he had difficulty in moving his eyes, particularly upward. He also noticed that vision became less acute and he tired quickly while reading. In January, 1917, he was treated at the Neurological Institute and later at the New York Hospital. A diagnosis of hysteria and later of cerebellar tumor was made at these institutions.

Family History.—The patient is the fifth

of six children. The patient's father is living and well at seventy. The mother was always nervous and had many attacks of hysteria practically all her life. She was always peculiar, fault-finding, very irritable and sensitive. She had a very definite paranoid trend directed towards her daughter E., whom she accused of attempting to injure her. She quarreled with this daughter for many years, and one day after a quarrel, at the age of sixty-six, she died from cerebral hemorrhage.

Of the six children one, A., is fifty-two. The wife and father and others who know this individual consider him a "boob." He is very cranky and has never done well at school. This man walks very unsteadily and "flops his feet about and swings his hands and body unsteadily in walking just like a drunken man."

C., aged forty-eight, is undoubtedly feeble minded. I have seen this brother and talked with him. He never did well at school. He has never been able to learn a trade, and now earns only \$2.00 a day "some days." He distributes cards and advertising matter, and until he got his recent job was often discharged for inefficiency. There are no trends or hallucinations or delusions, and he is best described by his own parent who says "he is like a big kid who has never grown up." This brother walks very steadily and shows no cerebellar signs. He is stooped, walking with his shoulders bent forward like an old man.

G. is forty-six years of age and the vice-president of a large bank. He has always been nervous and rather irritable. He has had two nervous breakdowns for which he has had to have treatment extending over several months. During the last year it is said that this brother is beginning to look like the patient and is "losing his mind." Recently he has had to give up work. There is no unsteadiness in gait.

E., aged forty-one, is the sister whom I have had the privilege of examining thoroughly. She has a very pronounced disturbance of gait with an ataxia limited

almost entirely to the right lower extremity. She has fallen many times while walking and has occasionally fallen or stumbled in her kitchen while crossing it during her household duties. It might be worth while to give a more or less detailed account of this patient. She is living with a man who is not her husband. She has had one child. She is very talkative, almost to the point of garrulousness, extremely irritable and boastful. She has no grounds for the belief which she has that she is very attractive and that many men are in love with her. When she was thirty-eight years of age, this patient began to notice that she was unsteady in walking, particularly in the dark. She noticed, too, that in going downstairs she would stumble and fall if she did not hold the banisters. Most of the trouble seemed to be in the right leg, which she said "flopped around as if it did not belong to me." On one occasion she went to light the gas in her kitchen late at night and fell to the ground. There have been no convulsions or periods of unconsciousness. For a number of years she had headaches, but they disappeared about a year and a half before the onset of her trouble. She complains of dimness of vision and has had to change her glasses several times during the past year. Her only child does not do well at school and behaves badly.

The neurological findings are as follows: There was nystagmus in both eyes on fixation to the right. The deep reflexes were 3 plus. The abdominals were all active and equal. There was hyperextension of both first phalanges and flexion of the second (both toes). There was a decided unsteadiness in walking and standing, the tendency being to sway or stagger to the right regardless of whether the eyes were open or closed. There was definite non-equilibratory ataxia in the right lower extremity with hypermetria. There was no adiachokinesis. Stewart Holmes's sign was present in the right lower extremity. There was a definite Babinski on the right side.

C. E., aged thirty-eight, is apparently the

most efficient of this family. He is the last born. Even he, however, has had difficulty in getting along. He has made a success in business and has had no disturbances of gait or of mentality. Nevertheless, he is rather quarrelsome, very obstinate and according to his parent and his sister-in-law "thinks he knows it all." He has been divorced from his wife.

There is no information about collateral branches of this family in respect to similar or other degenerative diseases. There is no insanity, epilepsy or other mental or nervous disease in any of its branches so far as the patient's father could remember.

VOLUNTARY MOTOR SYSTEM

Abnormal Attitudes and Deformities.—At the present time the patient sits huddled in his chair with a strong tendency to flexion forward of the entire trunk and of the head upon the chest. He will lie all day with his head bent forward, his chin almost resting upon the sternum without an attempt to raise his head. He makes strong resistance towards correction of this abnormal attitude. In sitting in his chair the head and trunk are also inclined to the right. His face is expressionless, and there is a narrowing of both palpebral fissures. He never looks upward or to either side. When the examiner comes into the room and demands his attention, the patient gradually moves his trunk and head backward until he can look directly forward. Movements of the eyelids and the eyes upward voluntarily appear impossible. The patient almost always sits with his legs crossed and makes rhythmical tapping movements of the heels on the floor. When asked why he does this he says that he does not know—"I guess I am nervous." If the patient makes any attempt to stand up, he falls immediately to the floor, almost always towards the right.

Gait.—At this time it is impossible to get him to walk. When I first saw him, however, there was a grossly ataxic gait, the patient throwing out his limbs in flail-like uncertain movements, the beginning of the movements being rapid, resulting in titubation. In walk-

ing, the normal associated movements were lost. There were irregular attempts on the part of upper extremities to make balancing movements associated with and compensating for the unsteadiness in gait. This was true also of the head. This disturbance of gait was not much worse with the eyes closed.

Co-ordination.—The patient is right-handed. He cannot stand with his feet together, eyes open or closed. There is a very definite Romberg at this time. When I first saw him, more than two years ago, he was able to stand quite steadily in the tests for equilibratory ataxia. He was then and is now, however, quite ataxic. There is gross ataxia, asynergia and hypermetria in the performance of the finger-to-finger, finger-to-nose, pointing and pass pointing movements and in all the tests to bring out these symptoms in the lower extremities. For instance, in doing the knee-to-heel test, regardless of whether the eyes are open or closed, the heel is almost always brought up to a point corresponding to the middle third of the thigh, and then uncertainly placed in the vicinity of the knee. The ataxia has always been uninfluenced by the equilibratory position and has been present regardless of whether or not the eyes were assisting the voluntary movements. There is marked adiadochokinesis.

Skilled Acts.—There is no loss of the motor formula for the execution of skilled acts. The patient was able to write fairly well when I first saw him, but cannot do so at all now. He grasps the pencil unsteadily, twitching and twirling his hand and fingers unsteadily towards the pencil and when the pencil is grasped there are wide, slow, irregular oscillations, giving one the impression of the intention type of tremor.

The patient talks thickly and slowly in a monotonous tone, the pitch at times being high and the voice and body partaking in no degree in the normal mimicry of speech. His face and his entire body is devoid of associated expression during speech. The speech is not ataxia. He talks very much

like an exhausted man who has absolutely no interest in what he says. There is no aphasia.

Abnormal Involuntary Movements.—When the patient attempts to do anything, such as reaching for a pencil or a glass, there is a slow, irregular, coarse oscillation in the entire upper extremity, particularly marked in the hands and fingers. When the patient grasps an object this tremorlike, athetoidlike movement is much improved, but in holding the object the oscillation continues, involving the entire extremity. There is no tremor of the face or tongue. The same ataxic, intention-tremor athetoidlike movement is present to a less degree in the right lower extremity and least of all in the left. Emotion increases the abnormal involuntary movement. There is none during rest and it does not appear to be influenced by attention. There are no movements during sleep. When the patient is particularly interested in making a special effort to grasp anything, his trunk and head and neck take part in the oscillatory movements.

Reflexes.—All the superficial reflexes, including the corneal, ciliospinal, abdominals, cremasterics and plantar, are active and equal, expressed by the numerals 2 equals 2. All the deep reflexes, including the jaw, pectoral, supra and patellar, biceps, triceps, radial, wrist, achilles and periosteal, are quite active and equal, expressed by the numerals 2 plus. The responses received in testing for deep reflexes are characterized by a slow muscular contraction rather than a quick jerk seen with the ordinary active reflexes. The test for the patellar reflex, for instance, causes a slow kicking up of the leg and a definite retarded resumption of the normal posture of the limb. This is probably due to the hypotonia which exists to a marked degree in this patient.

Muscle Strength.—The patient cannot rise from a recumbent or a sitting posture without a good deal of effort in which his whole body oscillates vigorously. He cannot stand alone. Contraction of the biceps and muscle testing of the triceps, flexors of the

wrist and extensors, also of the fingers and of the flexors and extensors of the thigh, knee and ankles and of the flexors forward and backward of the neck, fail to show any loss of muscle strength. The patient makes a good even resistance on both sides. In testing muscle strength, however, the normal rebound in the performance of Stewart Holmes's test is absent in all four extremities.

Muscle Status.—During the past two years there has been a gradual general mild atrophy which is part of a general loss in weight. There are no focal atrophies in any group of muscles. The muscles are soft in consistency and their tone is decidedly diminished. There is abnormal flexion and extensibility at all the joints of the extremities, most marked at the wrist and in the fingers. The hypotonia seems more marked on the right side, which probably explains why the patient's head and trunk tend towards this side when he sits huddled in his chair. There is no myodemia, and there is normal irritability to both mechanical and electrical stimuli.

Abnormal Associated Movements.—The abnormal associated movements have been mentioned in describing the gait and the tremor. They consist of oscillations in all extremities, trunk and head when voluntary movement is attempted. No other abnormal associated movements are present. When the patient laughs in his silly way, as he often does, his head rolls round and sways from side to side, and his extremities and trunk oscillate in the tremorlike movement also mentioned. His nerve status is normal.

GENERAL SENSORY.—All types of special and peripheral and deep sensation are normal. There is no agnosia.

CRANIAL NERVES.—All the cranial nerves are normal excepting the fourth pair and possibly the third. The patient's face shows an entire lack of expression both in repose and during speech. When he laughs or makes other involuntary movements of the face it is immediately seen that there is no

paralysis either of central or peripheral type of the seventh pair of cranial nerves. The eyelids are held in a semi-shut position and the patient does not seem able to lift them up entirely. There is almost surely a paralysis of upward gaze. Two years ago, when Dr. Holden examined this patient, he volunteered the information in the discussion that followed that in many cases of mental defect the patients had the subjective feeling that they could not look upward. Dr. Holden said at that time that all ocular movements were normal in this patient. Of late however, I have never been able to get him to look upward without his throwing his entire head and trunk backward to bring the object in line with vision. He never moves his eyeballs upward either voluntarily or involuntarily. It is with extreme difficulty that one is able to get him to look to either side but I do not believe that there is any paralysis of lateral gaze or any cephalogyric difficulty.

Vision is apparently normal in each eye. The fields as tested by Dr. Holden two years ago were found normal. At this time the patient complains that he cannot see as well as he did, but objective examination does not confirm him in his complaints. The fundi show nothing but a normal physiological pallor which was diagnosed in one of the institutions where he was seen before coming here as an optic atrophy. Dr. Holden says that his fundi are normal. The pupils are equal and normal in shape, in position and in their reaction to light, consensually, in convergence and accommodation. There is no strabismus and no diplopia. As mentioned above, movements of the eye upward are absolutely gone. There is no exophthalmos, Moebius or Von Graefe. The palpebral fissures are equally narrowed and appear to be in partial ptosis. There is no real nystagmus, but on fixation to the right, active nystagmoid movements are often seen.

Acoustic Nerve and Ear.—There is no trouble with hearing. Rinne's and Weber's tests are positive and the Barany caloric tests done two years ago gave the following

nystagmus reactions: With hot water the nystagmus away from the side stimulated continued for many minutes past the usual time. With cold water the slow movement of the nystagmus towards the side stimulated continued for sixty-five seconds. There were no irregularities of pass pointing. At the present time it is impossible to do these tests because of poor co-operation. There is however a spontaneous error of pass pointing of both extremities, the right towards the left and the left towards the right. Tests for this are not very satisfactory at this time, but with the right upper and lower extremity one gets quite constantly a wide deviation towards the left with the eyes either open or closed. A deviation is also present on the other side, but not quite so marked.

MENTAL STATUS.—The patient has completely lost interest. His memory is good. He recognized me when I called to see him after returning from overseas, recalled the occasion of our last visit and was able to tell when I last saw him. He gives with considerable accuracy the history of his trouble. He realizes that he is quite sick and is depressed. He is anxious for the end and has agreed to give his brain for study "if it will help anyone else." Despite this rather good retention of acquired memory, his lack of interest and his restricted environment caused by his disability have resulted in considerable dulling. He does not read any longer, is not interested by anything or anybody, spends his time nearly all day teasing and fooling with his wife's pet dog, whom he does not like, and finds much delight in annoying the dog into rages. He cries very easily for no sufficient cause. Often he laughs in a silly manner for hardly any reason at all. There is considerable lability of mood and much emotional deterioration. There are no delusions or hallucinations and no abnormal trends. Usually he is superficially depressed. He keeps in touch quite accurately with whatever business interests he may have. This condition of absolute loss of interest and emotional deterioration has

gradually become worse, until now he is content to sit huddled in his chair all day long tapping rhythmically with his heel and toe upon the floor. This apparently is his only form of amusement.

There is nothing abnormal in the cranial morphology, and the condition of the skin, nails and hair is normal. There are no visible or palpable glands. The heart and lungs are normal, the blood pressure is 128-80.

His general systemic condition is good. X-ray examination of his spine and upper extremities is negative. He has had a number of blood Wassermans which have all been negative. A complete spinal fluid examination was made twice at the Neurological Institute and once at the New York Hospital, and they were all entirely negative. The blood was examined for stipling of the red blood cells and no sign of lead poisoning was found.

SUMMARY

A disease beginning about the age of forty in a man who had frequent falls and many fractures of the upper extremities during an awkward boyhood. The patient has always been mentally below par. Onset with unsteadiness and ataxia, with an uncertain staggering gait and a defective speech and subjective eye complaints. The disease is progressive and undoubtedly familial. There are no changes in the reflexes, no disturbances of sensation, the blood and spinal fluid is negative. The fundi are normal. The Barany tests made two years ago showed hyperexcitability of vestibular functions. There are no cortical or peripheral types of sensory disturbance. There is no evidence of neurosyphilis. The diagnosis is progressive atrophy of the cerebellum, familial type.

The type seen in the patient's sister is not cerebellar and is more akin to the Friedreich complex.

In our patient the signs point to a more massive involvement of the right cerebellum and its pathways.

THE LIMITATIONS OF TINEL'S SIGN IN PERIPHERAL NERVE INJURIES

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Tinel's sign holds a valuable, though somewhat controverted position in the determination of nerve regeneration in peripheral nerve injuries. Its presence is taken by some to contraindicate surgical intervention and by others it is held to be of no significance. Neither view can be fully sustained. Careful consideration makes it apparent that with certain limitations the sign is of value. Its position in neurosurgery should be more fully appreciated, its anatomical basis better understood and its precise application to the clinical picture as a whole made.

As described by Tinel in the original article, *le signe du fourmillement* is the production, by pressure over the nerve trunk, of a sensation of electricity, referred to the peripheral cutaneous distribution of the nerve in question. The sensation is barely perceived at the point pressed, thereby differentiating it from neuritis, but is much more actively resented in the corresponding cutaneous territory. It is found within the nerve trunk when newly formed neuraxes are present. It is progressive along the course of the nerve in regeneration, and advances *pari passu* with the progress of the downward growth of the neuraxes. The exact amount of pressure to be employed, or the method of eliciting the sign, Tinel has not given. This may explain the varying technique and account for some of the discrepancies found by different observers. It is possible to make the sign more delicate by grading the pressure used so as to find the minimal threshold, and employing only sufficient stimulus to call forth peripheral tingling. Digital pressure may be used, or the nerve may be gently tapped with a small

hammer. Care should be taken to prevent spread and dispersion of the impulse. This may be minimized by resting the arm on a soft pillow and by an accurate appreciation of the course of the nerve.

According to Tinel, the presence of this phenomenon over the course of the nerve trunk, below the nerve injury, is not only a sign of nerve regeneration, but also an indication of the progress of regeneration. Its presence in a given case, if so interpreted, is thus a contraindication to surgical interference.

A study of peripheral nerve injuries both before and during operation cannot but cause amazement when it is found that though Tinel's sign had been definitely present, at operation, no apparent anatomical continuity could be established. Such findings are not isolated but are relatively frequent. It is this discrepancy which has led some observers to ignore the sign altogether. The tendency has been, perhaps, to depend too much upon this one sign alone, whereas the entire clinical picture should be considered.

In explanation of the presence of Tinel's sign in such cases, three main anatomical categories may be mentioned: (1). One in which there is definite continuity without an impossible barrier to the downgrowth of neuraxes, capable of producing a functional return. The neuraxes arise from the greater part of the central stump, are abundant, and follow suitable conducting paths. (2) One in which the anatomical arrangement is such that only a certain number of neuraxes may find their way into the distal stump, through and in spite of scar tissue, which, because of its very nature, would

tend to prevent an ultimate functional return. (3) Those cases in which even the remote probability of successful downward growth is precluded by the anatomical arrangement, for example, where the central end is widely separated from the distal end, the distal stump is turned aside, or back, or even adherent end-on to the adjacent tissues and without any intervening continuity apparent.

In all three categories Tinel's sign has been found. In all three it may be progressive along the nerve trunk. In the first instance the neuraxes are in sufficient numbers and proceed from a sufficient central source. In the second instance neuraxes are comparatively few, their source inadequate, i.e., coming from only a small portion of the central stump, and the tissue through which they pass is obstructive, making satisfactory regeneration improbable. In the third category a few scattered neuraxes may penetrate from adjacent nerve twigs, branches of the parent trunk, and injured at the same time, or from a widely diffused and dispersed downward growth, permeating in small strands along adjacent tissues, using them as a scaffolding, with only a very few finding the distal stump. Such downward growth we know to be possible from histological studies. Neuraxes may scatter widely and at great distances from the central end. But such growth, unless directed into proper channels, is abortive, destined to subserve no functional return. Yet these few neuraxes, when present within the nerve trunk, may give a positive Tinel's sign. It is impossible by this sign to differentiate such cases from those in which the downward growth is satisfactory and capable of ultimate regeneration. In both the sign may be progressive. Tinel's sign is qualitative, not quantitative. That the sign is due to the presence of neuraxes within the distal stump is evidenced by the fact that following operation and freeing of the nerve ends the tingling can no longer be elicited. Whatever continuity had been established is interrupted by freshening the nerve ends.

The following are selected from quite a number of cases to illustrate the discrepancy between the presumptive evidences of regeneration as manifested by the presence of Tinel's sign and the obvious improbability when seen at operation of a functional downward growth within the distal nerve trunk.

CASE I. Brief summary. Private S. Wounded by rifle bullet, July 30, 1918. Scar 8 cm. across middle of popliteal space with injury to both external and internal popliteal nerves. Date of operation March 13, 1919. Paralysis of all muscles below the knee with the exception of the outer head of the gastrocnemius. Mechanical excitability active in all paralyzed muscles. Electrical examination showed no response in any muscle excepting the gastrocnemius and the soleus—soleus 0.45 microfarads; gastrocnemius 0.4 microfarads. Tinel's sign present halfway down the leg over the anterior tibial nerve. It is absent in all other nerves of the leg. No evidence of contraction of the borders of sensory loss.

Preoperative diagnosis: Complete interruption, doubtful if anatomical, of the external popliteal; possibly complete in the internal popliteal. This differentiation was made, presumably, due to the presence of Tinel's sign, it being argued that there must be some continuity since the tingling of Tinel was present and progressive over the anterior tibial nerve below the level of the wound.

Operative findings: Internal popliteal nerve was bulbous, about twice its normal diameter. The nerve was lost in scar tissue. The nerve was exposed and traced downward through the scar. Its anatomical continuity was not lost, especially on its anterior part. The nerve was sutured end to end.

External popliteal nerve was completely divided with 10 to 12 cm. intervening. The central end was turned point on onto the biceps tendon and densely adherent. The distal end was small and lost in the surrounding scar. Multiple autogenous graft was done.

Unfortunately for the better completion of the record our artist was not available at the time this operation was done. The pathology here illustrates strikingly the improbability of any functional downgrowth with 10 to 12 cm. separating the nerve ends, the central stump being turned end-on onto the biceps tendon and the distal buried in scar. Nevertheless, Tinel's sign was not only present, but progressive over the anterior tibial nerve. This case exemplifies the third anatomical category referred to above, i.e., those cases in which the remote probability of successful downgrowth is precluded.

CASE II. Brief summary. Private R. Wounded by high explosive shell, July 26, 1918. Large scar over gluteal region with loss of muscle substance and injury to sciatic nerve. All muscles below the knee are



FIG. 1. SCIATIC NERVE INJURY, UPPER THIRD. Nerve completely severed as well as the nerve to the hamstrings. Note the wide separation of the nerve ends and the dense intervening wall of scar tissue, offering an impassable barrier to regeneration. In spite of the above anatomical arrangement Tinel's sign was present as far down as the ankle over the internal division.

paralyzed; only the semi-tendinosus contracts above the knee. Mechanical excitability shows good, quick, full response in all paralyzed muscles. Electrical examination gave no response up to 3.99 microfarads. Tinel's sign present over sciatic and down posterior tibial to 7 cm. above internal malleolus; none made out over external division. No evidence of sensory regeneration.

Preoperative diagnosis: Complete interruption of the external popliteal division of the sciatic; incomplete of the internal division.

Operation April 17, 1919. Complete

interruption of the sciatic nerve and lower branches to the hamstrings; gap of 5 cm. between the nerve ends with a dense perpendicular wall of scar tissue intervening. No apparent continuity to be made out. (See Figs. 1 and 2.) The distal stump was upon a more anterior plane than the central. Both the nerves to the hamstrings were sutured and also the sciatic.

The accompanying two drawings show clearly how impossible it would be for any adequate regeneration to take place. Yet in spite of the interval which separated the nerve ends and the intervening dense scar, some few neuraxes had penetrated, and Tinel's sign was to be made out over the internal division to within 7 cm. of the internal malleolus. Following the operation Tinel's sign could no longer be elicited, due to the fact that by freshening the nerve



FIG. 2. SAME AS FIG. 1. DISSECTION CARRIED ONE STEP FURTHER.

ends, etc., whatever continuity had been established was thereby destroyed.

CASE III. Brief summary.—Private J. Wounded by high explosive shell, right forearm with injury to the flexor tendons, ulnar and median nerves. Scar extensive and adherent. Tinel's sign present over both ulnar and median nerves as far down as the wrist. No other evidence of regeneration in the median. The ulnar showed some regeneration in the sensory fields. Mechanical excitability obtained only in the opponens pollicis. Electrical examination gave no response in any of the muscles of the hand.

Preoperative diagnosis: No regeneration in the median, some in the ulnar.

Operative findings: Median markedly

bulbous, continuity with the distal stump established by means of a small irregular band of scar tissue. (See Fig. 3.) This strand may have afforded passage for some neuraxes. The ulnar nerve was imbedded in scar; continuity not interrupted. The ulnar was liberated; the interval between the ends of the median, now 4.5 cm. after the ends were resected, was bridged by means of autogenous grafts.

In this case both the first and second anatomical categories, to which we have referred, are illustrated. In the median nerve the anatomical arrangement was such that



FIG. 3. MEDIAN AND ULNAR NERVE INJURY, MIDDLE THIRD FOREARM. Note the enlarged bulbous central stump and the tapering distal with a small irregular strand of scar tissue intervening. Tinel's sign was the only evidence of regeneration—obviously impossible of accomplishment. The ulnar nerve has been freed and the anterior and posterior divisions separated. Note the fusiform enlargement as contrasted with the large bulb in the median. Tinel's sign was present, as well as the progressive restriction of the borders of sensory loss as evidence of beginning regeneration.

a certain number of neuraxes might have found their way into the distal stump through the intervening scar tissue, but insufficient for regeneration of the distal segment. This is perhaps apparent in Fig. 3. In this case the median nerve gave a definite Tinel's sign. In the ulnar nerve Tinel's sign as well as the beginning contraction of the borders of sensation were present as evidence of regeneration. In this nerve there was continuity of the nerve pathway and without an impassable barrier to downgrowth.

Similar cases might be cited; however

these suffice to illustrate the points raised in this paper. In certain borderland cases the decision as to whether or not the downward growth is apt to become functional is a matter of surgical judgment, gained by seeing a large number of cases, and by a thorough study of the clinical picture as a whole. An intimate knowledge of the histological process of repair, such as is shown by Professor Huber, University of Michigan, in his exhaustive experiments in nerve regeneration, is of inestimable help in determining a correct operative procedure.

As seen by the above cases and by the anatomical considerations which they illustrate, Tinel's sign may be present and progressive in peripheral nerve injuries in which a functional nerve regeneration would never take place. However, a quantitative determination of Tinel's sign is not possible. Cases destined to complete regeneration may give the same response as those in which such return would never be possible. Hence Tinel's sign may be taken as evidence only of downgrowth of neuraxes irrespective of their quantity. The sign may be present when the downgrowth is abortive. Alone the sign is not evidence of functional regeneration but of the presence within the distal stump of young neuraxes—few or many. Therefore, it is not a sufficient contraindication to surgical interference. Unless this limitation is appreciated the presence of Tinel's sign may delay operative intervention. Even though present it should not prevent surgical assistance unless with it are other evidences of regeneration. If so considered and limited, it is then not only confirmatory, but it also enables one to measure the progressive downgrowth and advance of the process of regeneration. Its value is in conjunction with other evidences of regeneration; its danger arises when it is considered alone.

When the anatomical field is known, as in those cases who have been operated upon, it is of the greatest help as an indicator of regeneration and of its progress. Even here its limitations should be considered.

CONCLUSIONS

1. Tinel's sign may be elicited when regenerating neuraxes are present within the nerve trunk, irrespective of their quantity.

2. Such neuraxes may be found when a functional regeneration is not possible, due either to excessive scar or to dispersion and scattering of the neuraxes, so that only a very limited number may have penetrated the distal stump.

3. Tinel's sign, though present and progressive, must not be considered as evidence of satisfactory regeneration.

4. Tinel's sign is of greatest value when considered with other evidences of regeneration. When the anatomical field is known, i.e., following operations, it is of great help in following the progress of the downgrowth of neuraxes. Here its main limitations are removed, since the nerve ends presumably are so placed as to further proper downgrowth.

I take this opportunity to thank Lieut. Arthur B. Hamilton for his hearty cooperation and painstaking work.

A CASE OF PROGRESSIVE SPINAL MUSCULAR ATROPHY

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The progressive muscular atrophies form an interesting group in neurologic studies. There is quite a difference in opinion as to the differentiation of the cases and the location of the pathological processes. The spinal motor neuron is differentiated into three parts, the anterior horn cells, the motor nerve fibers and the muscle plate. Hoffman believes that the process is in the nerves, and proposed the term neuritic or neural form of progressive muscular atrophy; Bernhart the term "spinal-neuritic form of progressive muscular atrophy." Postmortem examinations by several workers, among others Marinesco, Sainton and Dejerine, have shown posterior and lateral column degenerations and atrophy of the anterior horn cells, anterior roots, the spinal ganglia and peripheral nerves. Some investigators do not attach as much importance to peripheral nerves as Hoffman does. Oppenheim in addition distinguishes a myositic form.

The question of fibrillation is an important one. Fibrillary twitchings are noticed under various circumstances—after excesses, when exposed to cold and in neurasthenic and hypochondriacal cases. They are then noticed in the small muscles of the hand, in the orbicularis palpebrarum and the muscles of the lower extremities. The higher grades of fibrillations occur only in spinal muscular atrophy. Fibrillations have not been noticed in the myopathies. Besides the changes in the anterior roots and the motor nerves, the muscles also share in the atrophy. They are pinkish or reddish yellow in color, and have fatty streaks throughout. Histologically, there is diminution in the size of the fibers, and the muscle substance is converted into granular and fatty material. The following case is typical enough to be differentiated from the various forms of

progressive muscular atrophy as one of spinal origin.

HISTORY.—The patient, J. M., forty-five years of age, single, porter by occupation, presented himself at this clinic on January 3, 1920, with the following complaints: diminution of the power of the left foot and leg; uncertain step, tripping easily; mild weakness of the right leg, at times suggesting to him involvement of that leg also. The onset of these difficulties dated back exactly two years, to January, 1918, and they gradually grew upon him. His first recollection of the trouble is the difficulty he had in going up a step ladder, when he would occasionally miss a step, and also stumbling in the dark, apparently over no obstruction. He believes the condition has been gradually getting worse. At present, he states, his left foot drops when he is walking and he can hardly raise it, i.e., flex it. He has tried to get on his heels at times and finds it impossible to do so with his left foot. He has no sphincter complaints, no weaknesses in the upper extremities, and does not complain about the rest of his body. He returned on January 31, 1920, with the added complaint that since the last examination pain had developed located at a midpoint in the lumbosacral spine, radiating down the external surface of the thigh on the right side; that now the right leg is surely also involved, since it has become weaker, with (in his own words) "its motor power slowed up." He feels now that he hasn't the strength in his legs that he should have for his age. He is sure the trouble is "creeping on him," making him gradually weaker. He is accompanied by his sister because he is continually afraid of falling. When this occurs he must get hold of something to get up because he hasn't sufficient strength in his legs to be helped by them alone.

The patient's previous history discloses nothing of value or abnormal. He was of normal birth, one of nine children. He was breast fed, always enjoyed good health, and to his knowledge acquired his teeth, began to sit up, walk and talk at the usual normal period. He recalls no diseases of childhood. He began school at six, and went through the grammar school. At thirteen he contracted diphtheria, which ran a moderate course with no complications or sequellae. He recalls no other sickness except an occasional cold. He never sustained any severe injuries, never was unconscious and never suffered from any motor or sensory disturbance.

The patient's family history is negative. There were no miscarriages. Three sisters and two brothers are living and well. One brother died at the age of one month of an unknown cause; another at fifteen years of typhoid; a third at twenty-five, of some affection of the lungs, its nature unknown to the patient. His mother died at fifty-six years of age from an accident, his father at forty-five from congestion of the lungs. All relatives, both on his father's and mother's side are well, none to his knowledge suffering from any disease of a similar nature, nor from any disease of a nervous or mental character.

The patient is a worried, apprehensive, middle-aged man, with deep furrows in the forehead and deep lines on the face. He was always a hard working man, began in life as a grocery clerk, and spent most of his life in the steel works, where he is at present employed. His work was first that of an errand boy; then in almost every capacity for short periods of time. He states that at no time was he in contact with fumes or the dust of metals; that his work was wrapping brass and iron beds in paper, or loading trucks; or when acting as porter, always resorting to wet sweepings. He does not believe that his work ever subjected him to noxious gases, poisons or dusts. He is careful in his habits, is clean and neat. He drinks coffee, but moderately, is a total abstainer from tobacco and alcohol. He does not use any drugs of any

description. Sexually he has been continent for the past ten years from choice. He denies any venereal disease, and does not admit the existence of any rash over his body, sore throat for a long time, or primary lesion. He sleeps well, has a good appetite, is regular in his alimentary and urinary habits; feels strong and sturdy except for his lower extremities and the nervousness over his condition, which lately has been constantly on his mind. He weighs 163 lbs.

EXAMINATION.—There are no abnormal attitudes or deformities that are discernible. In walking, there is noticeable the characteristic steppage gait of drop-foot. The gait is furthermore slowed down considerably, probably because of his subjective fear of walking. On his toes, his position is poorly maintained and for a brief time only. On his heels, he is unable to balance himself at all. Running is precluded, and Fournier's tests show the instability of the patient. The associated movements of the trunk, arms and head are normal.

Co-ordination.—The patient is right-handed. He performs the Romberg test fairly well. Standing on his right foot elicits a fair amount of balancing ability. On the left foot, it is difficult for him to stand. The finger-to-finger test is normal, as likewise are the pointing and pass pointing; the finger-to-nose, and each finger to thumb tests, eyes open and closed. In the lower extremities, toe-to-object and heel-to-knee tests were both normal. No adiachokinesis, dysmetria, nor dyssynergia were noticed.

Nothing abnormal was elicited in testing for skilled acts. He speaks in a firm clear voice, conversation is intelligent and sequential. His handwriting is rapid and rather bold, with no tremor or stops; there is no dyspraxia.

Of the abnormal involuntary movements noted there are quite a variety. There is a rapid course tremor of lips and fingers. There are very marked fine fibrillations all over the anterior surface of the

entire left leg. In some portions of the left leg there are fascicular contractions. At the second examinations fibrillations over the right leg were also noticed. The upper extremities, the anterior chest, and the back were entirely free from movement. The tongue showed a marked tremor on the right side.

Reflexes.—The jaw, pectoral, biceps, triceps, and radial were each evaluated at 2. The suprapatellar at first examination, $\frac{1}{2}$ right, $\frac{1}{4}$ left. Patellar, left $\frac{1}{4}$, right $\frac{3}{4}$. At second examination, one month later, suprapatellars and patellars on both sides were barely elicited tho slightly stronger on the right. The Achilles were consistently absent. The periosteal uncrossed, 1, right and left. No ankle clonus was elicited. The superficial reflexes were evaluated at 2 each—the cremasterics at 2+. Plantar flexion was normal on the right but not present on the left. No Babinski, Chaddock, Oppenheim, Gordon or Schafer reflexes were obtained.

The muscle strength test disclosed interesting variations, ranging from values 4 to normal. Rising from the recumbent position to sitting posture was well performed, to standing more clumsily. Testing the dorsi, flexing power of the left foot determined its value at 4, i.e., the patient was just able to lift and sustain the foot in a dorsi flexed position. The head, shoulders, arms, forearms, hands and fingers, in all planes and at all angles, disclosed no loss of power. The scapulae in the shoulder and arm movements were normal. The trunk was perhaps slightly diminished in strength. The thighs on flexion, extension, abduction, adduction, rotation inwards and rotation outwards showed a strength less on the left than on the right, the latter likewise perceptibly diminished. The left leg on flexion and extension was very much diminished in strength, perhaps about 20 per cent, and the right less than that. The feet on plantar flexion and dorsal flexion showed the right but slightly involved, the left markedly. Abduction and adduction was a little better performed than the above. The toes on flexion,

extension, abduction and adduction on the left showed practically no power, and on the right diminished power. There seems, then, to have been a marked involvement of the peroneal and anterior tibial group of muscles involving markedly on the left, partially on the right, the sartorius, supplied by the crural nerve, and the adductor muscles supplied by the obturator (L 2, 3, 4). Flexion and extension test of the thigh disclosed the affection of the tensor fasciae femoris and the gluteus maximus supplied by the gluteus superior (L 4, 5, S1) and inferior (L 5, S1, 2) respectively. Also the Ischiadic nerve (L 5, S1, 2, 3) supplying the obturator internus, the gemelli, and the quadratus femoris which perform the external rotation; also the biceps femoris, the semi-tendinosus and the semi-membranosus which perform flexion of the leg. Of the muscles supplied by the peroneal, as shown by inability to extend the toes, there were involved the tibialis anticus, which performs dorsal flexion and supination of the foot, and the extensors of the toes, also the peroneal muscles which perform dorsal flexion and pronation of the foot. The nervous tibialis likewise must have been involved, as shown by the inability to plantar-flex the foot, involving the nerve supply to the gastrocnemius, soleus, tibialis posticus and the flexors of the toes. The atrophy was not very marked. On mensuration the left thigh was $17\frac{3}{4}$ inches, the right $18\frac{3}{4}$; the left calf $14\frac{1}{4}$, the right calf $14\frac{3}{4}$. The consistency of the muscles was soft in most parts of the lower extremities. There was lack of tone on the left, slightly diminished on the right. The fibrillary twitchings were more noticeable on the left than on the right. Eliciting the knee jerks started up more than the usual movement of the muscle fibers, tending to show marked mechanical irritability.

The electrical reaction showed sluggish faradic, normal galvaine, response on the right foot and leg faradio gone and galvania diminished on the left. The sum total of the electrical changes in the lower extremities

showed a reaction of degeneration on the left and a partial R De on the right. There were no abnormal involuntary movements.

GENERAL SENSORY.—Sensory examination tested over both upper and lower extremities showed acuity, localization and discrimination normal and equal on both sides. There was noticed nothing abnormal in testing sensibility to pain, both superficial and deep. Cold and warm temperature tests disclosed no abnormalities. Vibratory, pressure, muscle, tendon sense, stereognosis, and barognosis were normal.

CRANIAL NERVES.—The olfactory nerve was found normal. There was nothing abnormal noticed in the examination of the nose. The vision, tested by the eye department, was slightly diminished right and left. The fundi were negative. Visual fields roughly tested, were normal.

The pupils were 3.5 mm. in size, were equal, circular, and centrally placed. They reacted to light and convergence normally. There was no Argyll—Robertson pupil, no hippus, no strabismus, and no diplopia or other deviations from normal noticed. The movements of the eye were normal. No nystagmus was noted. The palpebral fissures were normal in size and equal. No ptosis, von Graefe, Kocher, Stellwag or Horner signs were present. The acoustic nerve and ear were found normal. The fifth nerve was found normal with the corneal reflexes present on both sides. The mouth showed no malformations of the arch. Only seven upper and seven lower teeth remained. These were cavitated and decayed. The gums showed marked pyorrhea and the trauma of recent extractions. Taste was normal. The movements of the upper and lower facial muscles were normal. Nothing abnormal was noticed in the vasomotor or secretory status. The glosso-pharyngeus and vagus nerves were normal. The position of the uvula was normal. The pharyngeal or palatal reflexes and voice were normal. Swallowing and respiratory movements were normally performed. The eleventh nerve was intact. The position of the tongue

when protruded was normal as was likewise its length and shape. There were marked fibrillations more particularly on the right side of the tongue. Its movements were normal in all directions. Articulation was normal. Nothing abnormal was noticed in the trophic state of the tongue.

MENTAL STATUS.—The patient was of fair intelligence, attentive, possessed of good memory and was emotionally stable. He suffered from no compulsions, had no hallucinations or delusions.

SYSTEMIC.—The integument, hair and nails were normal. There were no pigmentations or other surface peculiarities. The left foot was somewhat colder than the right. There were no secretory or vasomotor disturbances except cyanosis of the left foot as compared to the right. There was no dermatographia or Sargent's line. The patient possessed no abnormal palpable glands. His heart rate was 72 per minute and was of good character; no murmurs were heard. His blood pressure was rather low, 110 over 60. The respiratory, gastrointestinal and genito-urinary tracts seemed to be negative. The urine report showed a clear, acid, amber colored urine. The specific gravity was 1020. No albumin, sugar, indican or bile pigments were present. On microscopic examination only a few urates and occasional leucocytes were found. The Wassermann report on the blood is negative.

DISCUSSION

The case presents a few points of unusual interest. The usual peroneal type of tooth generally occurs late in childhood, while in this case we have the onset late in life. No etiologic factor is here ascertainable. No history of any toxic or noxious product, of trauma or exposure to severe chills can be obtained, nor has it been possible to establish any hereditary factor. Whether the case can be differentiated from chronic anterior poliomyelitis is left open for discussion. The history of paralysis of the peroneal group of the left foot two years ago, insidious in its onset, followed by atrophy of the calf

muscle and subesquent involvement of the right foot, the reaction of degeneration complete on the left and partial on the right, the atony, the vasomotor symptoms of cyanosis, coldness and lowered blood pressure, the absence of reflexes or any abnormal involuntary associated movements, the fibrillary twitchings, and the absence of the Babinski, and doro-pedal reflex, point to the involvement of the anterior horn cells. The differentiation of the case from progressive muscular dystrophy lies, among other points, in the preference the latter has for the proximal muscles and those of the trunk, in its hereditary or familial nature, its occurrence

in youth, etc. It is distinguished from multiple neuritis by the absence of sensory symptoms of ataxia, of the history of known noxae or the psychio disturbances characteristic of that disease; from gliosis, syringomyelia and occupational atrophy by the absence of sensory and sensory diasociative symptoms. It is differentiated from amyotrophic lateral sclerosis by the absence of the Babinski phenomena; by the absence of tendon reflexes, and by the lack of spasticity. The above findings and the course of the disease suggest the diagnosis of a spinal form of progressive muscular atrophy of the peroneal type.

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ACUTE INFECTIOUS MYOCLONUS MULTIPLEX AND EPIDEMIC MYOCLONUS MULTIPLEX*

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Preliminary Report

The acute infections often show a tendency to become localized in this or that portion of the nervous system, the explanation for which is far from satisfactory in the present state of our knowledge. For example, in acute poliomyelitis, or as it is now commonly known, the Heine-Medin's disease, we recognize a number of clinical types which are dependent upon the seat of localization of the toxins, or other infective agent in the tissues. There is a cerebral, spinal, pontine, bulbar, cerebellar, neuritic and meningeal form of this disease, the same infective agent producing very different types of clinical reaction. Certain forms, more especially the spinal, are not uncommon as sporadic manifestation, while in epidemics all types are frequently encountered. It would, therefore, appear as if certain strains of the same organism may have special elective affinities for certain tissues of the nervous system, and that in this way there are produced the special clinical types of reaction which have been referred to. Other factors of a theoretical nature may also be considered, as, for example, the exhaustion or biological inferiority of certain neural systems which render them more susceptible to the deleterious influences of toxic products. Be this as it may, the clinical reactions to infection often follow certain defin-

ite types, which it is important to recognize, from the standpoint both of diagnosis and of localization.

As *Acute Infectious Myoclonus Multiplex* and *Epidemic Myoclonus Multiplex*, I would direct attention to one of these special forms of localization in an acute infection of the nervous system. This form is characterized by lancinating pains, muscular contractions and twittings, and a delirium of toxic origin. This group of symptoms, in my experience, constitutes a well defined clinical type of neural infection, quite different in certain essentials from those previously recognized, and may be encountered both in *sporadic* and *epidemic* form.

SYMPTOMATOLOGY

The onset of the disease is acute, and is characterized by sharp shooting pains in the trunk and extremities, at first local but rapidly becoming generalized. They may reach an extreme degree of intensity and become truly agonizing. As a rule the pains continue with diminishing severity over a period of one or two weeks, but rarely may persist with considerable intensity for three or even four weeks. Spinal pains are also sometimes present, but are not nearly so intense as in the more peripheral areas. The pains are soon followed by the characteristic muscle jerks, waves and twittings (myoclonus

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multiplex, myokymia, and fibrillary contractions). The contractions follow in the wake of the pain and make their appearance first in those parts where the pains were first manifested. In some cases an interval of a week may elapse before the appearance of the myoclonus and myokymia. The muscular twitchings are bilateral, multiple, and may be generalized. A tendency to localization in certain regions of the body may also occur, and more especially in the trunk and the abdominal musculature. The contractions are quick and of short duration, involving individual muscles or portions of muscles, but not synergic groups, so that the resulting locomotor effect is comparatively slight. When severe, slight movements of the toes, fingers, and extremities may occur, but never to the same degree as in chorea or cortical myoclonia. The muscles of the lower extremities and trunk are particularly susceptible, and especially the abdominal musculature and the posterior muscle groups. The myoclonus and myokymia may disappear after two or three weeks, but in one case of the sporadic type, the muscular twitchings continued for several weeks. In fat subjects the muscle undulation and twitching may be readily palpated when otherwise somewhat concealed by a thick layer of adipose tissue. In one of my cases, an extremely obese woman, with marked delirium, which terminated fatally, the presence of slight muscle waves and twitchings could be felt in the abdominal walls and calf muscles which were not visible beneath the heavy panniculus adiposus. In some cases the facial muscles are slightly involved as well as the muscles of the tongue, and the articulation under these circumstances may show slight involvement. This could not be dignified by the term dysarthria, but nevertheless there is produced a slight inconstant disturbance of the nicety of articulation, due to the slight wave-like undulations in the facial and hypoglossal musculature.

The above symptoms are accompanied by moderate fever, which varies from 100° to 101°F. In severe and fatal cases the temperature may rise in the later stage of the

disease. There is also a distinct acceleration of the pulse rate, and a pulse rate of 100 to 120 may accompany a moderate elevation of temperature.

In all of the cases there is a delirium which varies in duration and intensity with the activity of the infection (*myoclonus-delirium*). During the more active period of the disease there is often marked hyperhidrosis, and the degree of sweating seems to bear some relation to the activity of the myoclonic phenomena. There is no tenderness of the nerve trunks, but during the painful period there is general sensitiveness to movement and surface impressions. The myotatic irritability of the affected muscles is increased and there is also a quite definite exaggeration of the normal myoidema (idio-muscular response). There is no paralysis or paresis of any muscle, or group of muscles, and no anesthesia. There is no ataxia and no loss of the deep sensibility. In two cases during the first few days of intense pain transient paresthesias were felt in the feet and legs. These, however, were of short duration and disappeared with the pain. The tendon reflexes are present and active. In only one case, late in the disease, the Achilles jerks were not elicited, and the knee jerks were somewhat diminished. The cranial nerves show no evidence of involvement, other than the myokymic twitchings noted above. The optic nerves are normal. The skin reflexes are present and equal (no Babinski). When abdominal myokymia is present, the abdominal reflexes are exaggerated. No sphincter disturbances were noted, and no herpes zoster. The spinal column is not tender on percussion. There is no rigidity of the neck, and Kernig's symptom is absent.

In brief, the clinical picture consists of lancinating pains, myoclonus and myokymia multiplex with delirium, the whole course of the disease suggesting an acute infectious process with a special involvement of the central nervous system. The duration of the disease varies from six weeks to two months. Convalescence may, however, be prolonged, and in one patient the delirium

with myoclonus continued for three months.

Eight cases of this unusual affection have come under my observation during the past sixteen years, the first in 1904, and another ten years later, in 1914. During the past three months I have seen six cases of this curious form of infection. The two early cases are of the sporadic type; the later cases belong to the epidemic form. The characteristic muscular twitchings appeared, in all of these cases, a few days to a week after the onset of the disease, and were of the same character in both the sporadic and epidemic forms. They consisted of partial and total contractions of individual muscles with but little locomotor effect. The contractions are involuntary and involve individual muscles which as such are not under the control of the will. All degrees of contractions were noted, the whole muscle, portions of muscle, and the individual fasciculi (*myoclonus multiplex*, *myokymia multiplex*, and *myoclonus fibrillaris multiplex*). Synergic groups of muscles were never affected, as in convulsive seizures of cortical origin. Spinal puncture was performed in four of the cases, in three the results were negative, in one there was a moderate pleocytosis. Blood culture was taken in one case, and was found negative. The urine showed traces of albumen and hyalin casts during the acute stage of the disease in four of the cases.

There were no general or focal cerebral symptoms save the delirium which was present to some degree in all of the cases. Headache, vertigo, and convulsive seizures were absent.

The *myoclonus delirium* presents the characteristics of a toxic delirium with fleeting hallucinations, illusions, and transitory delusions. There is restlessness, insomnia, fear and apprehension, disconnected thought and mental confusion. In the severe type in the later stage, there may be apathy and a tendency to stupor. The mental symptoms are all more conspicuous at night, and the delirium content frequently takes on an occupation form. In the graver cases there was a distinct attention disorder with symptoms of Korsakow's syndrome. This was dis-

tinctly present in two. Of the five cases which survived, three made complete recoveries and two are convalescing. Convalescence was quite prolonged in one instance.

DIAGNOSIS

The peculiar nature of the motor disorder which these cases present is well recognized in neurological literature, and is described under a variety of headings. There are, for example, the paramyoclonus multiplex of Friedreich, the myokymia of Schultze, the myoclonus fibrillaris multiplex of Kny, and the so-called fibrillary chorea of Morvan. These are generally regarded as representing only different degrees of involvement, all corresponding to a fundamental type, and referable to the lower motor neurones. They may be produced by a variety of causes, among which may be mentioned mental and physical shock, exhaustion, exposure to cold, diabetes, lead poisoning, trauma, alcoholism, and they may follow sciatica and poliomyelitis. Similar muscular phenomena have also been observed after certain infectious diseases, e.g., diphtheria, malaria, and acute articular rheumatism.

In the cases which are here described the mode of onset, the course of the disease with fever, accelerated pulse and delirium, are indicative of an infectious disease. It must therefore be regarded as an obscure type of neural infection which is expressing itself by a peculiar and characteristic train of symptoms. I would also emphasize the fact that there was no essential difference in the cases which were of sporadic and those of epidemic origin.

Multiple neuritis may be excluded by the absence of weakness or paralysis in any group of muscles, the absence of objective sensory disturbances or loss of reflexes, and especially the freedom from tenderness along the nerve trunks. *Acute poliomyelitis* may also be excluded by the absence of paralysis and consecutive muscular atrophy. *Dubini's Disease*, an obscure affection of the nervous system, which is endemic in certain parts of Italy, notably Lombardy may, I

believe, also be excluded. This affection in its more acute form is characterized by headache and backache, and is followed by myoclonic contractions of the cerebral type (whence the term "chorea electrica"), also by convulsive attacks resembling Jacksonian epilepsy. Later there is paralysis of certain muscle groups which had been the seat of myoclonus, in some cases with wasting and changes in the electrical reactions. It is an obscure form of neural infection which may occur both in acute and chronic forms. The sensorium is, as a rule, free. So far as I know the disease has not been observed in this country, and yet a possible relationship to the epidemic forms of encephalo-myelitis should receive serious consideration.

The *Epidemic Encephalitis* or *Lethargic Encephalitis* should be especially considered in this relation, as it has reached epidemic proportions at this time and may present itself in a variety of forms, cerebral, spinal and neural. What I have described as *epidemic myoclonus multiplex* probably belongs to this group, and represents a special "*myoclonus type*" of this affection. This cannot be determined at the present time with certainty, as we do not know the infectious agent of either disease. It is probable, however, that the infectious agent of epidemic encephalitis and epidemic myoclonus multiplex are one and the same. Additional weight is given to this theory by the coexistence of cranial nerve palsies and myoclonus in undoubted cases of epidemic encephalitis.

The lancinating pains, the myoclonus and myokymia in this group of cases are unquestionably referable to the lower motor and sensory neurones. In a general way the localization may be said to be in the spinal cord or somewhere in the course of the peripheral nerves.

The nature of the lesion and its more exact localization are as yet purely speculative. It is clear that the toxin has a selective irritative action upon both the peripheral motor and sensory neurones, with a resulting functional disturbance, but without evidences of distinctive or degenerative effects upon the neurones.

The interesting and unusual feature of this group of cases is that in spite of a severe and sometimes lethal infection of the nervous system, the motor and sensory symptoms are irritative in character without evidences of paralysis or anaesthesia.

This, so far as my experience goes, is a unique manifestation and is not encountered in other forms of myelogenous and neurogenous infections. The occurrence of the myoclonus symptom-complex in sporadic form would also show that we are dealing with a definite "clinical type" reaction, which in the uncertain state of our knowledge concerning the cause and localization of myoclonus multiplex may best be designated as *acute infectious myoclonus multiplex* (*epidemic myoclonus multiplex*). The delirious form may be characterized as myoclonus-delirium.

AIMS OF BIOCHEMISTRY OF THE NERVOUS SYSTEM

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Our present notions of the chemical constitution of nervous tissue as a whole, and of its single morphologic elements, are not very far-reaching; the knowledge of its constitution in pathological states is barely outlined. This is also true of the chemical modifications occurring in nerve cells and fibers when they are the site of disease processes. We know still less about the chemical and physicochemical changes which take place in the various functional states of these elements, and little about the way a stimulus is transmitted through them and into what form of energy it transforms itself in order to be transformed into consciousness as a sensation, or in generating a glandular or muscular reaction.

Nevertheless, our researches in this field have already contributed considerably to the progress of our science, and they present themselves to our younger experimenters as the most promising of all methods of arriving at the solution of so many questions that occupy the biologist in general, and the neurologist in particular.

The major value of these researches, however, is in having extended to those special tissues and those biological phenomena the elaborate methods and the physicochemical laws which are the basis of general science, thereby giving a new and promising direction to a branch of morphology and physiopathology which hitherto has found so little nutrition along the hackneyed routes of anatomy, pathological anatomy, experimental and clinical physiology.

The latter sciences by means of the intensive inquiries and brilliant discoveries of the latter decades have built a solid basis for a theory of the nervous system, both as to its minute structure and also as to its

functional differentiations and histological alterations in the various nervous and mental diseases. But that substantial analytical work has exploited to-day its field of study to such an extent that it can add very little else by adhering to the same methods of research. It is indeed a fact that every branch of study has a limit of perfection in its technical means of investigation and in their evaluation and interpretation; beyond which it cannot proceed to new conquests without equipping itself with new means furnished by other branches.

Let us briefly consider the various sciences which interest and concern our special field of study. The macroscopic anatomy of the nervous system, both descriptive and histologic, has now reached such a stage of progress in the vertebrates and in man that very few of its last secrets are still unveiled. Histology has become an ever keener investigator, and has already squeezed out from the thousand and one methods it uses in its researches all that they could possibly yield. It has been a most valuable help to physiology and pathology; but it is quite safe to say that histology also has reached the limits of its potentiality, for up to now it has had to avail itself largely of technical artifices which alter the tissues and bring into relief structural images which we have every reason to believe are in most cases very far from the real morphology of the living cell. Hence that feeling of uncertainty and perplexity which seizes the student when he considers the entity, or lack of it, of certain structures described in the nerve cells and neuroglia fibers and cells, as well as the physiological deductions which have been drawn as to the function of such structures. At any rate it is to this science, as well as

to macroscopic pathological anatomy, that the most solid foundations of our knowledge of clinical neuropathology are due; and a great deal is also owed to it by psychiatry, which is toilsomely constructing its field and seeking a framework of material facts for its precious array of clinical observations. But histology itself must have new means at its disposal to bore deeper into the mystery of intimate cellular constitution. Having so far rested upon images artificially produced by means of laborious attempts, it must needs become more rigorously scientific, in the sense of knowing the material upon which it operates, evaluating the processes of extraction, precipitation, and elective coloration which it employs in its technical proceedings; of ascertaining, in fine, upon what substances, or group of substances, it works while speculating upon the structure and form of those substances. Hence the necessity of suspending for a while these efforts which are so tiring, for they have never up to yesterday seemed to reach the goal. The necessity is felt by many neurologists, psychiatrists, physiologists and biologists, of seeking new resources by analytical means and informative concepts of the general sciences which deal with the phenomena of matter and energy under whatever forms they appear.

The advances made by general chemistry during the last few years, and especially in the field of colloidal substances; the discoveries of physical chemistry which have had so many applications in biology and enlighten at present all modern physiology, were naturally destined to find a field for application even in the studies of the constitution, composition, structure, functions and energy exchanges of the nervous system. And indeed during later years there has been no lack of pioneers in this new field of labor, nor is there a dearth of their persevering successors. Let us then see briefly whether these attempts were justified, and whether these broader horizons of an otherwise rigidly experimental science justify the aspirations of these pathfinders.

The biochemistry of the nervous system

encompasses the study of the chemical composition of nervous tissue, and the physico-chemical phenomena which it develops. In so far as the nervous system is a substance to be analyzed, it interests the chemist as much as the biologist, while in the domain of energetic phenomena it interests the physicist, the physiologist and the pathologist. We notice, in fact, that chemists like Thudichum, physiological chemists such as Ostwald, and physicists like Nernst, each deal with the subject with objectives purely chemical, physicochemical and physical; while for the biologists the ultimate objective is by this means of research to clarify the problem of form and function of the elements of the nervous system, in its normal and pathological aspects.

This distinction between the two objectives responds to the different mentality, or rather mental preparation, of the present students of experimental science, inasmuch as biology has not yet reached that degree of exactness that will gain it admittance among the physico-mathematical sciences. But progress towards this event is steadfast and continuous. It is barely a century since Lavoisier and Laplace, by announcing that heat which animates the living organism results from a combustion similar to that of our hearths, laid the basis for a fundamental truth ever more triumphant since then in the succeeding advance of experimental sciences: i.e., that the physicochemical processes which rule living beings come within the ordinary laws of physics and chemistry. It is therefore to be expected that within a few decades biologist, physicist and chemist will combine as one in the study of the body. For the time being, let us take notice of this happy collaboration among students, which will ultimately lead to their unification, and see what fruitful labors they prepare for the branch which is now our main interest.

The chemistry of the nervous substance has put in evidence a quantity of compounds which in part are common to other animal and vegetable tissues and in part are peculiar to the nervous tissue proper. The first difficulty that presented itself in this sort of

research was that of differentiating the chemical products really existing as such in the complex admixture of organic and inorganic substances that make up the tissue; a difficulty, this, which is common to all similar researches conducted upon other tissues, animal and vegetable. Here also it was necessary to proceed by steps, that is, by separating the groups of substances from which, through selective processes, it was expected to obtain the single pure products. Since the very first researches, which hark back to a century ago (Vauquelin and Couerbe) the high percentage of fatty substances existing in the brain was recognized, and it was attempted to extract them fractionally by aid of the usual solvents of fats. But it had to be admitted later that some of these solvents—as for instance ether—can alter the substance they dissolve by oxidizing it; while others, like alcohol, dissolve too many in the solution, making it difficult to separate without altering them. Other means, as certain salts greedy for the water of crystallization—can easily dehydrate the lypoideo-proteid mass, but will make it quite difficult to obtain from such a dry mass the pure extracts of those salts. As we weigh the merits and demerits of so many means of fractional extraction recommended as a satisfactory aid to the analysis of the nervous substance, we find that to-day the method most preferred is the one advanced by Sigmund Frankel. This is primarily designed to find a process of fractional extraction which will make it possible to separate the various lipid products of the brain without a modification of that composition which they presumably exercise upon the living tissue. In our laboratories, where for several years we have been experimenting with several of these modern analytical processes, we have repeatedly found the Frankel method more practical than others (especially more than that of Koch and Mann which is based on alcohol); so much so that it could be relatively easily applied to the examination of various brains, both healthy and diseased, with satisfactory results. I shall illustrate what further re-

searches may be expected along these lines by the use of this method.

The nervous tissue—brain, marrow, nerves—cleaned of the pia mater and the blood by means of simple water spray, is run through a meat chopper, weighed and passed successively through the following lipid dissolving substances:

(a) In acetone, which, being soluble in water, dehydrates the mass in the first place, and in the second, extracts the cholestrin and a first group of non-saturated phosphatids;

(b) In petroleum ether (boiling below $55^{\circ}\text{C}.$), which extracts the non-saturated phosphatids and the cerebroses;

(c) In benzol, and

(d) In alcohol, both of which extract the remaining cerebroses and the saturated phosphatids;

(e) In ether, which extracts the lipid residuum.

By these successive extractions, the mass, reduced to powder after the first dehydration with acetone, is freed from the water, the salts and extractive substances and from all its lipid components. What remains is a mixture of proteid substances.

By this process it has been possible to confirm and enlarge on what previous experimenters had brought forth by various means; *i.e.*, that the cerebral mass is made up of a high percentage of water (77%) and what remains of solid substances is made up of $2/3$ lipid and the other third of proteid substances. The prevalence of lipid substances in the nervous mass, and their high biological significance as shown by so many physiological and physiopathological researches, rightly leads us to consider such substances of peculiar importance for the structure and function of the elements of these organs. Hence so much labor to isolate, to differentiate them, to define them chemically and to know their physicochemical properties.

By the above described fractional extraction we obtain first a general rough separation of the lipid groups, such as may serve as a measure of comparison, the formula of the percentages resulting being constant for

every species and every segment of the examined organ.

Thus in the single fractions, it is possible to differentiate the single components ultimately. This is not the place to go into details. I shall confine myself to pointing out what now are generally admitted as the most firmly established facts in this field, in order to indicate the roads opened for such researches to future workers.

In the first acetonic portion there pass, besides the water which is mostly removed by the first acetonic extraction, the whole of the cholesterin and a great deal of the non-saturated phosphides. It is a well-known fact that the nervous substance, unlike the blood, bile, and other tissues, does not contain any cholesterin united to fat acids in ethereal form, but only free cholesterin, which represents about 10 per cent of the solid substance (Bunz, Frankel). Among the mixture of phosphides which constitute almost all the rest of the acetonic extract, the largest component is a deca-amino-di-phosphatid, isolated and analyzed by H. Elias in Frankel's laboratory and named "leucopoline" on account of its being equally distributed both in the white and gray matter of the nervous tissue.

The ethero-petrolie extract which follows represents by itself almost 28 per cent of the dry mass and two-fifths of the total lipoids; it completes the extraction of the phosphides non-saturated, of which the chief representative is a mono-amino-mono-phosphatid discovered by Thudichum himself, Parnas and Neubauer and named cephalin. Other phosphides which are not yet well identified chemically, pass into this fraction, but with a much less quantity of cephaline. Only one among these, very much like ovoidecthin in its chemical and physicochemical properties, was isolated by Frankel and Linnert and named sahidine. This is a tri-amino-phosphide, precipitated by cadmium salts from the alcoholic extract of the etheropetrolie fraction, and is probably the same compound which was mistaken for lecithin by Thudichum. This latter substance, according to recent

analyses, does not exist in the cerebral mass, or, at least, it has not been found there with our present means of research.

In the above mentioned etheropetrolie extract and in the subsequent extracts: benzolic, alcoholic and etheric, other lipid substances appear, of which only a few have been identified so far. In the group of the saturated phosphides the best known is the sphingomyeline, isolated by Thudichum, which contains phosphorus and sulphur. Another very important group is also differentiated here, made up of nitrogenless compounds and called cerebroses, or sphingogalattoses, because like the common glucoses, they contain in their molecule a hexone which has been identified in the galactose. The two cerebroses that contribute the highest percentage to the brain are the phrenosine and the cherosine, studied and isolated by Thudichum and more recently by Thierfalden and Rosenheim. These two substances seem to be really pre-existent as such in the tissue. It is admitted to-day (Posner and Gies, Frankel) that the former identified itself with the body which Thierfelder isolated and called cerebrone and which is made up of a molecule of galactose, on the basis of formula $C_{17}H_{35}NO_2$ (a bivalent aminalcohol called sphingosine) and an acid not yet well specified which is called phenosinic acid, but which is not a stearic acid, as Thudichum contended. The cherasin differentiates itself from the phrenosine by a different acid which it contains identified with lignoceric acid (Rosenheim and Levene). The two cerebroses are represented in the brain in the proportion of 2 per cent of the fresh substance.

These are the lipoids which chemical analysis of the nervous substance has so far identified with sufficient certainty. They are the highest percentage of the tissue; but many other lipid substances still remain in the different extracts, of which some have been incompletely analyzed, and others have not yet been separated in the rough.

In the proteid residuum three different globulins have been identified (Halliburton), nucleins (Jaksche and Geoghegan)

and a sort of aminoacid well studied by Abderhalden and Weill and by our own school of Bottazzi.

Among the extractive substances of especial importance are the purin derivatives (0.024 to 0.092% of the fresh substance), the galactose (0.40%) which Jaffé calls the sugar of the brain, and the inosite (0.19-0.22%).

With this research material at hand the students of cerebral chemistry attempted to approach the question of the differentiation of various parts of the tissue. Thus appeared the first comparative analyses between the gray substance and the white; between the brain, the cerebellum, the bulb, the medulla oblongata, the medulla spinalis and the peripheral nerves, etc., and comparative chemistry was extended to the various ages of the individual and to the various species. From the total of these researches it seems to result that in man and the higher animals the components of the nervous substance so far chemically known, are met in all the segments of the examined tissues, while, on the contrary, their percentages vary. It has not yet been possible to establish whether certain substances particularly belong to determined sections of the nervous system or to specific species. So far the specificity of belonging to the gray substance and not to the white seems to be admissible in the case of but one body, *i.e.*, a sulphurated nucleoprotein isolated by Halliburton and analyzed later by Levene and Koch. The other substances chemically known were found in all parts of the nervous system, but with noteworthy changes in their quantitative percentages. It is thereby proved that between the white masses and the gray, between segment and segment of the cerebro-spinal axis, there are quantitative differences of the components of the tissue, the possible qualitative differences remaining still inappreciable.

I shall only recall some of the most important characteristics. The gray substance is much poorer in lipoids than the white; it contains less cholesterol, less phosphides, very much less (about 10 times) lipid sul-

phur; while it contains a larger quantity of proteins, nucleoproteins, neutral sulphur and 14 per cent more water. The white substance, on the other hand, contains much neurokeratin which seems peculiar to the myelinated nerve fibers (Koch).

The pons, bulb and medulla are, in comparison to the brain, very rich in non-saturated phosphides, while they are poorer in saturated phosphides; the medulla being the part of the neuron richest in lipoids, and among the latter the richest in non-saturated phosphides. In addition, its chemical composition appears to vary according to its tracts. The interesting researches of Buglia and Maestrini, which are well deserving of being continued and extended, show noteworthy quantitative differences between the various fractions of nitrogen and phosphorus in the dorsal and ventral cords.

As to the phases of development, the neuron gradually loses its water percentage while it gains in lipid substances. Among these, the cholesterol and the non-saturated phosphides increase, the saturated phosphides decrease and the cerebroses accumulate progressively in the fibers following the process of myelinization. (According to Smith, Mair and Wlassek the Weigert stain is due to the presence of cerebroses in the fibers, while according to Reich it is entirely due to lecithins. Furthermore, between individuals we find quantitative differences between the lipid groups better differentiated; these differences are constant, especially in the medulla [Dimitz].)

The results of these studies, especially those of experimenters who have applied the most searching analytical methods, such as Linnert and Dimitz, are therefore the particular chemical constitution of each segment which, according to several authors, allows us to suspect a differentiation still more characteristic and minute of every stratum, every bundle or tract and every nerve.

Aside from the purely chemical interest in these studies of nervous tissue, they acquire a vast significance in the hands of the biologists, for they tend to a direct

knowledge of the material of which that tissue is woven and of the manifestations of energy which issue therefrom. With the first purely chemical objective, we aim to construct the rational basis of histological technique which is entrusted with the discovery of the minute structure of the brain. Histology has always attempted to get at the reason of its technical manipulations and the images it displays through them. Whether it proceeded along the lines of fixation and inclusion, or colored and differentiated directly the fresh tissue, or applied the more recent physical methods, direct and indirect (ultramicroscope, polarizer, lights of least waving length, etc.), it has always aspired, in the case of its most noted students, to straighten out the chemical and physicochemical processes which complete its proceedings, and estimate what it exactly does when it fixes and precipitates substances; when it uses solvents and extracts, when it corrodes and colors this rather than that; when it sees and does not exactly know what it sees. The truth is that so far it has lacked the elements to get out of its empiricism, and has had no time to wait for them by giving up in the meantime the great problems of fine anatomy and pathology of the nervous system. Its triumphs have, indeed, been most brilliant, but lo! it has reached the dead line where "doubt springs out at the very feet of truth."

It must needs now go back over a good deal of its road, winnowing its results in the wind of facts which chemical analysis is gradually raising. But already new paths are opening before the tireless wayfarer. The histochemistry of the brain acquires every day a fresher impulse and a speedier momentum, and it gets where it can, where it is permitted to arrive by the still too limited progress of the general biochemistry of that tissue. It strives by means of elective colorations to set in evidence this or that chemical compound, this or that mixture; it tends to differentiate the lipid substances which appear in the neuropilic and perivascular nervous elements when the organ is diseased; it seeks to sur-

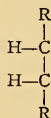
prise those substances during the processes of autolysis in the experimental degenerations, and it applies to microscopic sections the processes of fractional extraction with lipid solvents (Reich, Buscaino); it uses upon them the differentiative optical means in comparison with the pure substances extracted from the nervous mass. Much empiricism is still extant here, but the pathway is blazed towards a perfection ever more scientific and exact by means of research and the element of judgment. The parallel progress of these two branches—researches within the substance as a whole and within the differentiations of the components in the histological element—are preparing the material for observations upon which some day we may found and describe a normal and pathological chemical anatomy; *i.e.*, an anatomy aware of the material subtraction of appearances which will bring evidence by no longer using empirical artifice, but one of true chemical reactions, or of optical reactions to the various lights.

The second objective concerns the energetic properties of isolated chemical substances and their participation in the physiological processes of the tissue, which is essentially *biochemistry*. It aims at the discovery of the elementary physiology of the organ, going from the simple to the complex, rising from the properties of isolated substances to those of their mixtures, of the structures that those mixtures form, of the histological elements which result from those structures and of their relations to one another and the ambient liquids, such as electrolites, enzymes, hormones, etc.

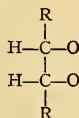
The substances referred to above have already been honored by a rich literature which refers to their chemical, as well as chemico-physical and biological properties. The readers of this review are already too conversant with the numerous researches on the properties of cholesterin and phosphides in general for me to dwell at length on this subject. That the cholesterin, which is so largely represented in the brain, plays even here its part of fixer of the phosphorylated lipoids and aid to the saturation with water;

that it electively fixes there toxic products either extraneous or hormonal, and that it contributes towards the maintenance of the tension of the solvent is quite probable, as many researches tend to prove. This substance, which I shall refer to later in connection with the pathological chemistry of the brain, is the most stable component of the nervous tissue, in that it remains at a high percentage even when other lipoids have undergone lytic and diffusive processes.

Particular attention should be paid to the properties of the cephalin and the sulphurated lipoids. Cephalin is the major representative of the non-saturated phosphides of the brain, and as such it possesses a double chain of its carbonic atoms:



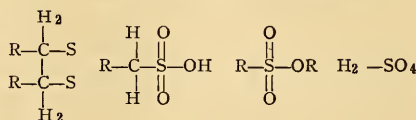
To this double chain, which is very frail, it owes its property of energetic reductive (as it may be observed by ordinary methods), and of being able to absorb in its make-up much molecular oxygen:



The oxyhemoglobin of the arterial blood can, therefore, yield to it its oxygen, which, on the other hand, can be utilized for other oxidations by means of ferments and special reactions.

Another group of bodies which seem to participate in the respiratory exchanges of the tissue is that of the sulphurated proteids and lipoids. By becoming oxidized the proteid sulphur becomes neutral sulphur (a compound that has in its molecule bodies analogous to taurine); through a subsequent oxidation the latter is changed into lipid sulphur and finally to inorganic sulphur

(Koch) according to the following table:



All these various fractions of sulphurated compounds may be—according to the process of extraction of Koch and Mann—isolated from the nervous substance and valued in their percentage in the various parts (white and gray) of the tissue. It is indeed significant that it was just on the fractions of neutral sulphur, sulphides non-saturated and phosphides, that the few researches so far made in the field of pathological chemistry have made evident the great differences from the normal, which allow one to suppose a concomitant alteration in the oxidation of these substances in the diseases investigated. The latest researches on the oxygen interchange in the nervous system—differing from previous investigations—agree in establishing the fact that this organ has a respiratory exchange which is very active in the waking state (Gaida, Winterstein) and that especially in the cord and the bulb the consumption of oxygen exceeds, in proportion to their weight, that of any other tissue (Winterstein). Given the high caloric value and the avidity for oxygen of the non-saturated phosphatids and their existence in higher percentages in the cord and bulb, given also the great reductive and oxidative properties of the sulphurate compounds, it is easy to attribute to these substances a noteworthy part in internal respiration of nervous tissue.

I will touch in passing upon other important properties both physico-chemical and biologic of the components of the nervous mass; for example the absorption and solution of many organic and inorganic substances aside from the non-saturated phosphatids, which permit them to assimilate and select certain circulating bodies, and thus participate in the interchange between tissues. I shall remain longer, however, in

consideration of the problem of the assumption, fixation and the chemical annexation of an elective nature of toxins, poisons, narcotics and excitants. This property has recently interested many experimenters who see in it a promising means of interpretation for the pathology of many diseases. Such researches have given us the view that the cerebral substance behaves in a vastly different way in its reactions to the bodies and compounds which result in physiological or pathological changes. For instance, in regard to toxins, it may neutralize or enhance their action, may absorb in indeterminate quantities or fix them strongly. It is still a matter for discussion as to which component of the brain—cholesterin, frenosin or a protein—is the fixing or the neutralizing agent of the tetanus toxin; or if cephalin phosphide fixes and enhances the toxicity of the diphtheritic toxin, malleinic acid and tuberculosis, what or which are the components (lipoids or ferments?) which so rapidly kill the tubercle bacilli? But the facts of the fixation, neutralization and the exaltation of the toxic actions exist, and it will be the reward of future work to clear up these problems. Analogous to this are the promising announcements of the elective actions of other substances. For example, lead, which has a predilection for the ganglion cells of the bulb and the pyramids (Camus) strychnin for the anterior horns, cocain for the posterior horns (Toraco Sano), the anaphylactic poisons for the bulb (Achard and Flandin), collargol which fixes certain fibers (Axenfeld and Maestriani), the toxins, viruses, certain hormones, certain anesthetics, which elect territories peculiar to them (one thinks of primary degeneration of the lateral columns in a number of toxic processes; arsenic, phosphorus, diphtheritis toxin, and in parathyroidectomy); all these facts tend to show us that each segment of the nervous system reacts in an elective manner to certain substances, and that this in turn modifies itself physiologically.

This electivity of various regions of the nervous system for certain toxic agents explains better than anything else the state of

our knowledge concerning many pathological processes producing the various system diseases. One thinks of the extreme dilution of certain substances which may affect the nervous system, because of the simple fact that they have this power to fix themselves to certain determinate elements—for example aconitine, which in 3 or 4 milligram doses kills a man, because from the general circulation it fixes itself upon a few ganglion cells in the medulla.

The laws of chemical affinity, and of the chemico-physical modifications by contact, of solution, absorption between reacting bodies, also apply to the various components of the nervous system and its fluids. As a result of the thought that each part of the nervous system has its peculiar chemical analysis, it is obvious that any circulating substance in the blood or spinal fluid which bathes the entire nervous system, will react only on those portions for which it has an affinity either chemical or physicochemical (meaning by the general term affinity those complex processes which preside over the reaction between substances.) The method of reaction may vary with individual substances, or with the same substance in various places; the reaction may be purely chemical—as with the oxidation of the non-saturated phosphides; or chemico-physical—as with the absorption of toxins by catalytic processes or the modification of tension or the dialytic constancy of the liquid when determined lipoids pass into solution; or physical—as the depolarization of cells when special agents modify the potential of their membranes. But all these reactions are always chemical and energetic and show the elective tendency for certain tissues, by localization topographically in certain organs. These are the conclusions which the above-mentioned studies lead us to, and they show us with a new light the pathogenic mechanisms of the alterations which clinical and anatomical pathology produce in nervous and mental diseases.

In a great many of these diseases—whether they are caused by trauma or by vascular and meningeal lesions or by tumors,

the factor of intoxication is the most important. Even in infections the toxins determine the type of nervous or mental disease; and even where the infection injures directly the nervous tissue, as in tabes and paresis, or glanders and tubercular meningitis, etc, it is more to the diffusion of the products of exchange in the invaders than to the organisms that one must attribute the grave symptoms and the relative histologic lesions. As to many diseases we know only in part the nature of the toxic agent, as in uremic convulsions or diabetic coma; as to others we know nothing, as in manic-depressive psychoses, dementia precox and epilepsy; or in nervous lesions Freidreich's disease or spastic spinal paralysis and multiple sclerosis. It is likewise true that even here one deals with toxic products of necessity which show an affinity for certain elements of the nervous system, as the histologic evidence conclusively shows. Their origin is not important whether they come from outside the nervous system or are manufactured within it. Many of these substances have an exogenous origin, many probably come from within the tissues. It is certain for example that with nerve tumors, even small ones, certain destructive syndromes may follow which Lugaro attributes to the factor of auto-intoxication from neuroglia proliferation—probably due to a perversion of its antitoxic properties. To an analogous alteration, biochemical and secretory, one might ascribe the symptoms of epilepsy, linked as they are to gliomatous lesions of the brain of a circumscribed character.¹

These are the interesting conquests left to the pathology of the future, to isolate these imagined toxic substances, establish their origin, bring to light the relations between these agents and the chemical constituents of the various parts of the brain which react

to them, and determine whether it is true that physico-chemical laws governing their elective reactions are controlled by the coefficient of partition between the two phases of aqueous-lipoid, and whether one deals with absorption caused by diminished superficial tension of the absorbing medium (according to the thermo-dynamic law of Gibbs) and not really with a series of chemical reactions.

* * *

The chemistry of the brain, however, has brought some direct contributions even in these fields, limiting itself at present to diseases in which there are changes in the cells and nerve fibers, in which differences have been found in the qualitative and quantitative chemical constituents of the nervous mass. These are the first attempts to differentiate between the pathologic and normal brain, which show a trace of the great possibilities of chemical pathology of many diseases which will prepare for the more arduous task of the chemical histopathology.

I shall only touch upon the most important results of these researches. The mental and nervous diseases which have been studied so far from this viewpoint are the following: dementia precox, dementia senilis, progressive paralysis, pellagra, amaurotic idiocy and hemiplegia due to cerebral origin.

In two cases of amaurotic idiocy (infantile) histologically studied, Dr. Mott, with the collaboration of Sidney A. Mann, conducted the chemical research, extracting the brains with alcohol and ether and determining from the proteic residuum the nucleoproteids (proteid phosphorus) and the simple proteids. Having histologically ascertained the disappearance of the Nissl substance from all the cells of the gray substance and a great increase of neuroglia cells, he found by chemical analysis a great diminution of nucleoproteids and a not small increase of simple proteids. Mott interprets these two findings as being respondent to each other, concluding that the substance which forms the Nissl bodies is a nucleoproteid, while the neuroglia cells are made up of simple proteids. This would find

¹ This conception deserves a larger illustration which I propose to work on later on the basis of much material collected during the war, including the most varied epileptic syndromes which followed cerebral injury often insignificant in nature.

confirmation in the fact recorded above that only in the gray substance—and not in the white—or, at least, in a negligible quantity—do we find sulphurated nucleoproteids. As the difference between the white and gray substances lies in the presence of nerve cells in the latter the hypothesis naturally arises that the nucleoproteids are peculiar to these cells and precisely to the substance contained in them which manifests itself in the Nissl bodies.

Dementia precox was studied by W. Koch and Sidney A. Mann by modifying and improving the proceedings referred to above. In nine brains, they found compared to what is normally found, a marked constant diminution of the neutral sulphur, while the proteid and inorganic sulphur are relatively augmented. From the above consideration of the probable oxidizing processes which underlie and are controlled by the sulphurated products of the brain, the two English authors drew the inference that in such a disease a noticeable deficiency of those processes is found, and they put in relation these findings with the added neutral sulphur found in the urine of these patients, which is very marked, especially in the initial and acute stages of the disease (Pighini). Recently I have worked upon eight brains affected with dementia precox, following the Frankel method which I have adopted in my laboratory in order to evaluate comparatively the findings derived from various diseases. From the data gathered so far—and which I propose to publish shortly—there appears a constant diminution of all the lipid fractions, except that of cholesterol. The Eteropetrolie fraction is not as low as in progressive paralysis (see below), the most diminished being the benzolic and alcoholic fractions. The brains of the precociously insane, would then seemingly lack more especially the saturated phosphides, sulphides and cerebroses. Researches now going on concerning the content of phosphorus and sulphur in the various extracts will throw additional light on the question. To the decrease of lipids corresponds an increase of water.

In the case of progressive paralysis we have, so far, the researches of Koch and Mann, mentioned above, and those of Smith and Main and those of Carbone and myself which correspond to and complete each other. The first two English authors found a decrease of lipid phosphorus in five cases; Smith and Mair noticed in three cases a deficit of 50 per cent of cerebroses; while Carbone and myself found a decrease of all lipid fractions and especially of those which comprise the non-saturated phosphides. Here also cholesterol remains in normal proportions, if not in excess, and here also, as in the case of dementia precox there is an increase of water and an increase of the total proteids. The increase of water in such brains was noticed also by Barrat.

As to pellagra we have a diligent study by Mathilda Koch and Karl Voeglitz (1916) conducted along the methods of Koch and Mann. These American authors have established parallel researches upon the brains and the medulla of monkeys and rats kept on special vegetable diets (maize, raw turnips) and of pellagrous men—and they found upon composite chemical analysis by means of normal controls the same quantitative modifications, to wit:

I. A tendency to increased water-volume, a loss of lipids and a slight tendency to decrease of protein.

II. A decrease of cerebroses, phosphides and sulphides probably due to an accentuation of the lipolitic processes and to degeneration of the tissue.

III. A relative increase of cholesterol in the cerebellum and the spinal cord.

IV. A slight diminution of cholesterol in the brain.

V. The proteins seem to be the least compromised among all constituents of the nervous mass. They maintain the normal percentages in the brain and the cerebellum. In the cord there is noticed a decided increase relatively to the dry tissue, while relatively to the fresh tissue there is a diminution.

VI. A considerable increase of the extractive substance (increase in the ex-

tractive colloidal nitrogen) which could be in relation with the pathologic decomposition of certain hydrophile colloids and in relation to the decrease of the latter.

VII. A loss of neutral sulphur in the brain and the cord and an increase of it in the cerebellum—to be interpreted probably as an alteration of the oxidizing processes of the colloidal sulphurated compounds.

As to senile insanity, which ought to offer an interesting field of inquiry in relation to the extended and characteristic histologic alterations offered by the brain, we only have the incomplete researches of Allers. This author, using the extractive Frankel process has published so far only the results of acetonic extraction in three brains of seniles, proposing to continue the analytic study of other extracts. His researches, mayhap, were interrupted by the war. Allers' finding, however, is interesting because of the fact that in the acetonic extract, besides the cholesterin and leucopoline which, as we have seen, pass normally in that first fraction, he has brought out another phosphide, but saturated, which does not give the V. Bayer reaction and containing in its molecule the group of galactoses (it gives the reaction of orcine). It would seem then that it is due to the abnormal presence in acetonic extract of a phosphogalactosid having the property of dissolving in acetone, differently from the normal phosphogalactoses which only pass in the successive extracts—benzolic and alcoholic. Allers rightly considers this body as a product of the decomposition of more complex lipid bodies—or lipoproteid—caused by the pathologic process; and he presumes that it could be identified with those products of demolition which histochemistry reveals in such brains as these (Alzheimer) and which are precisely soluble in acetone.

I remember some fragmentary researches on the cerebro-spinal axis in hemiplegics, and of nerves in Wallerian degeneration. In the brain and cord of hemiplegics in the fresh state there were found an increase of water and a diminution of lipoids excepting

cholesterin, which was present in normal percentages (Smith and Mair, Mott and Bar-rat). In dry tissue the cholesterin is greatly increased in relation to the other lipoids. Even in nerves showing Wallerian degeneration we find a diminution of phosphorated lipoids, which is caused by a substitution of "fats" deprived of phosphorus for those lipoids which react to osmic acid (Marchi). This reaction is characteristic of fats with a double chain of carbon atoms, and it is probable that the phosphides not saturated become split off during the degenerative process and the phosphorus molecule is eliminated. Only the non-saturated fatty acid remaining. Besides the lipoids phosphorated, the cerebroses are diminished; to this is due the loss of the Weigert stain in degenerated tracts. An increase of water and corresponding degeneration of lipoids is found in the central nervous system of dogs affected by rabies (Udransky).

Ciacco found a diminution of phosphides in brains of pigeons fed on polished rice.

The chemical analysis of the pathological nervous system, as we have seen, has barely been touched upon, and these investigations have been done by different methods, few of them similar. Notwithstanding, considered as a whole, they offer significant results, and they may open the way to the demonstration of how effectually certain degenerative processes can be discovered in the elements of the nervous system.

Histology has grossly indicated the images of cells in chromatolysis, of bundles in degeneration both secondary and primary, the accumulation of disintegrating substances in nerve cells, neuroglia, and adventitia, and has showed them in the most varied types of pathological processes. But concerning how the cell and nerve fiber and other neurological elements become diseased, histology has been unable to differentiate by any given pathological process or with any toxic agent. The complex anatomical pictures macro- and microscopic, and the site of the lesions, have been well defined anatomicopathologically in many mental diseases, like general paresis, senile dementia, arterior-

sclerotic dementia, cerebral syphilis in its varied forms; but the examination of one section under the microscope, one ganglion cell in chromatolysis or a prey to lipolytic processes, one giant neuroglia or amoeboid cell of any form, does not tell us whether the brain belongs to a paralytic or dementia precox, to an epileptic, a senile, a case of rabies or one of glanders. There are, it is true, some few characteristic things, like Fischer's plaques in senile brains, the rod-cells which are seen in paresis, the tortuosity and reduplication of vessels in seniles; but these appearances seem linked especially to disintegrative processes in the nervous substance and its fluids, and they give testimony as to the severity or intensity of the breakdown. The nerve cell elements follow in disease, a constant line, regardless of the different causes which produce it; the line runs from the state of reintegration to swelling, edema and the change in the Nissl bodies and passes on to the graver alterations of disintegration—lipoprotein, lipolysis, and the break-up of run through the pictures of the so-called primary degeneration and secondary degeneration (Schmaous, Pighini, Perusini) following always processes which are more progressive towards fat. The results of chemical analysis throw light upon these histological findings. One fact comes out prominently and is common to all the morbid processes examined; that is the characteristic behavior of the aqueous content, cholesterin and of the lipid mass. Calculating the percentage as regards fresh tissue, in all pathological conditions so far examined there has resulted a quantitative increase of water and a diminution of the lipoids, while cholesterin remains hardly changed.

In all the diseases with a degenerative course, nervous tissue tends to become impoverished in a certain class of lipoids and rich in water. Because it has not been definitely demonstrated that such brains do not weigh less than normal, or at least the proportion between cranial capacity and the brain (as calculated by Reichardt and Apelt 100.88 cmc.) is not diminished, it is easy to suppose that the water has taken in part

the place of the lipoids which have disappeared. The diminution of these varies qualitatively and quantitatively in various diseases, as undoubtedly it varies in various cases of the same disease. In the evaluation of the analytic data one must bear in mind many factors, such as the age of the subject, the individual variations; and as I have pointed out, the percentages of the various components of the brain vary with age, and especially the white matter presents variation in different individuals (Linnert). The variations from the normal in the above-considered examinations are too great to be considered other than pathological. And so we see in general paresis that all the lipoids are diminished, which are extracted with petroleum ether and benzol, alcohol and ether, with the predominance of the phosphides, non-saturated, (cephalin), while in dementia precox this fraction is spared in comparison with the other saturated phosphides, cerebrosides, and sulphides; the same finding is encountered in pellagra and in degenerative processes of the fibers of the nervous system. To such losses, in all these diseases, is the great contrast seen in the conservation *in situ* of cholesterin. I would like to add that the cholesterin remains unchanged even in arterio-sclerotic processes even when the other lipoids have disappeared.

This is the general line of the chemical processes of a degenerative nature in the nervous tissue which have already been discovered even with the few and various methods of approach used in investigating these diseases. Along these lines it is quite possible that quantitative differences may be demarcated between the single diseases, but at least they indicate the way which must be followed to reach the light. These chemical processes of progressive type in nervous tissue merit a great deal of attention from the biochemist. To interpret them one must place himself on the practical ground of the acquired ideas of the physical chemistry of the various lipoids, and of the method by which they participate in the structure of the cellular bodies. I shall touch only lightly on this interest-

ing question. Researches have recently brought to light a fundamental difference between the chemico-physical behavior of cholesterol in relation to the other lipoids of the nervous system. Cephalin, lecithin, the cerebrosids with the organic solvents (chloroform, alcohol, ether, carbon sulphide, etc.), do not form true solutions, but act as do the hydrophilic colloids in their relation to water. Cholesterol, however, does not show this colloidal-like relation to these solvents. I would remind you of these facts, viz.: substances soluble in organic solvents—such as analine, benzoic acid, naphthaline, camphor—raise the superficial tension of these solvents, just as the organic salts raise that of pure water. Cholesterol behaves like these substances, while the phosphatids and the cerebrosids lower the superficial tension of their means of dispersion, whether it is water or organic solvents. These last lipoids dissolved in chloroform, instead of increasing its volatility diminish it, whereas cholesterol raises it notably. This occurs equally with the weight of the molecule, a phenomenon which is not seen with the other lipoids (only frenosine in alcoholic solution—probably because of its faculty of crystallizing out from the alcohol—shows this character of molecular solution in respect to the pressure of vapor). Other important properties characterize the difference between these two groups of lipoids as regards the phenomena of absorption and mixture. It is known that the presence of one lipid influences the solubility of the other, and how their coexistence in cellular protoplasm in the proportions necessary to molecular solubility, or in the phase of colloidal suspension, or in liquid crystal form, whether it is above all influenced by the physico-chemical reciprocal action between these substances or with other constituents—proteids, salines, etc., of the protoplasm. It may at this point be noted that in these complex actions cholesterol behaves in a physico-chemical manner which in more than one regard differentiates it from the other lipoids above mentioned. The experiences of S.

Loewe are interesting apropos of this question.

Methylene blue is not soluble in tetra chloride of carbon. If however there is dissolved in this a minimum quantity of lipid, for example cephalin, and if the colored substance is sprinkled on the liquid—the methylene blue will dissolve immediately. To clear up this action of the lipid the theory of the partition of colors in a biphasic system is advanced as follows: Lipid—chloroform water—methylene blue.

Varying the initial concentration of the color in its aqueous phase, one obtains for each lipid a characteristic “isothermy of absorption” represented by the curve $\frac{dx}{dc}$

in which x indicates the quantity assumed by the chloroform phase, and the equilibrium of concentration of the color in the water phase. If, now, instead of varying the concentration of the color one varies the volume of chloroform, and with that the concentration of the lipid in the chloroform itself, one finds that the measure of absorption of the color is entirely independent of the chloroform portion of the lipoido-chloroform, as is demonstrated by the curve of cephalin. Therefore absorption takes place only in the lipid portion, and one deduces that the lipoido-chloroform solution must be micro-heterogenous, containing therefore the lipid in the form of active colloidal particles which are in suspension. This is the behavior of the cephalin, of the cerebrosides and of the other brain lipoids. The cholesterol however follows another curve of absorption, demonstrating how on the one hand this least favors the assumption of the color, and on the other hand showing how that assumption is independent of its concentration in the chloroform. From these facts it is added that the lipoids of the first group manifest a tendency to a colloidal dispersion even in organic media (and therefore differing from the hydrophilic colloids like albumin, being called anophilic colloids). While in lipoids of a second group (timolo and linseed oil) to which

cholestrin belongs, the degree of dispersion in organic media is independent of their concentration, as is found in the aqueous solution of soap. Because of this quality of the anophilic semi-colloids it is proposed to call them "semi-lipoids."

I have referred to the varied behavior of the two lipid groups—lipoids and semi-lipoids—in order to place in relief the differences manifested in their behavior as regards degenerative processes of the nervous system. These processes are concerned chiefly with the lipid components of the nervous system, or at least the quantitative alterations of these components are those which, until now, have been the most obvious; and they are confirmed by other than chemical examination, by the histo-chemical, for the extensive lipid degeneration which is seen in all these nervous diseases is seen in the tissues under the microscope. This last fact must be considered while we are commenting upon the chemical analysis. I may be permitted to mention the researches of Barbieri and myself on the comparison between the chemistry and histo-chemistry of nervous tissue in general paresis, and the considerations discussed relating to the apparent contradiction between the chemical analysis and the histologic findings. In the microscopic sections, especially of certain areas (like the frontal and cornu ammonis) the lipid products in nerve cells and mesodermal cells are abundant, and seem to consist in the ganglion cells mainly of non-saturated phosphides (method of Ciaccio and Smith, myelinic droplets, anisotropia). However in the glia cells and mesodermal and in the subpial spaces, it consists mainly of phosphides, cerebrosids and cholesterin; while these same phosphides—except cholesterin—in many of those brains present a percentage much lower than in normal brains. This double row of facts was interpreted as the effect of an autogenous-lipoid degeneration of the tissue, and finds some analogy in the production of aseptic autolysis (Fontanesi, Ziveri, Biondi, Simon) even though here the process, the cycle being suspended, was

accomplished under entirely different circumstances. The mixtures of proto-lipoids in ganglion cell protoplasm, which give during sleep the histo-chemical reactions of lipoids, because of the regressive pathological process undergone, release their component lipoids, part of which diffuse out of the cells and are mobilized from the nervous and mesodermal elements, in order to be later thrown back into the circulation—according to the processes presumed by Alzheimer; part—the cholesterin—remaining *in situ*. And by this means we explain the apparent abundance in the tissues of those lipid compounds colored by the elective histo-chemical methods.

The different physico-chemical behavior *in vitro* of these two groups, lipoids with a colloidal function and semi-colloidal in organic solvents, find therefore a contrast also in their different behavior *in vivo*, when they exist in a tissue like the nervous system of which they form a large part. Modifications in their physiological equilibrium occur just as may happen with disease.

The arduous problem is presented of interpreting the process *in vivo* upon the slight evidence which we have of the behavior of these processes *in vitro*.

If the cerebro-spinal diseases so far considered have demonstrated chemico-pathological alterations of a uniform nature, it is easy to suppose that an analogy of action resides in the pathogenic causes which produce them. If these causes are identical in special toxic agents—which may be admitted for many reasons—they must be attributed to the chemical and chemico-physical action of these toxins, and their degenerative effects. But our opinions in this regard are very limited because it has not been possible to isolate even in heterogenous mixtures any of these supposed toxins. As regards paresis we know of the existence of the spirochete in the brain, and it is therefore obvious that the syphilitic toxin, even more than the microbe, plays its part in the morbid process. In dementia precox and pellagra we have nothing but the theory of the alterations in exchange, and of vitamin

or endocrine defect, all of which tend toward a conception of auto-intoxication. And even in the processes following trauma of the nervous pathways by means of focal areas of destruction due to hemorrhage or direct trauma causing Wallerian degeneration, one may admit that an endotoxic action exists in these nervous pathways which are isolated from their trophic centers, being thus deprived of the conduction of their physiological stimuli and of the oxygen exchange which accompanies this function.

Why, in view of these obscure morbid agents, do the lipo-proteid and lipid substances undergo these regressive changes? The grave problem must be faced in various directions. One may formulate many hypotheses, each one of which must be illustrated experimentally. Two of these are:

(A) That the pathogenic agent alters definite diastatic actions in the nervous elements. We know that these tissues are rich in ferments, especially oxidizing agents (Batelli e Stern, Rosell, Pighini, Juschtchenko, Ciacio) which make certain the play of catabolic processes in the protoplasm. Their equilibrium being altered, these catabolic ferments, ilpoproteid and lipolitic, may predominate. Researches concerning ferments in the pathologic state are completely absent.

(B) The pathogenic agent splitting off into the aqueous and lipoproteid emulsion of the protoplasm, becomes attached to certain lipid components of the protoplasm by means of a chemico-physical analogue of the above described biphasic system, namely, the lipid-chloroform methylene blue water. We have seen that each lipid follows an isotonic method of absorption of its own. Therefore this would explain the breaking down of the equilibrium as a result of which these substances are found in the cell bodies. Each phase of dispersion reacting in its own specific way as regards the substance absorbed, this would tend to a collection of homogeneous drops, and to a differentiation of them from their medium. In this way they become visible to histochemical examination. Concerning the diffusion or not of these substances in their liquid environment,

and their being encased within glia cells and mesodermal tissue, the chemical and chemico-physical properties of each colloid play their own part, and in this way the final process is brought to light. We hold at present that the phosphides manifest the greatest tendency of any of the cerebral lipoids to form emulsions with water, and Novi has demonstrated *in vivo*, obtaining a marked diminution up to half of these lipoids in dogs which had had distilled water injected in their carotids. Cholesterin however remains *in situ*. To these two facts Novi attributes the psychic excitement produced in these animals.

We hold that they do not occur in the physical state as colloids in the myelin sheaths of the nerve fibers, but as liquid crystals, and that they reach this state by the assumption of some water as water of crystallization.

The experiences of Reich, and especially of Gothlin, are interesting. Minimum quantities of water are necessary to obtain the myelinic form of the non-saturated phosphides of the brain (cephalin and lecithin). Adding to this however an excess of water one gets a disappearance of the crystalline form of the liquid, and the lipid diffuses itself into the colloidal phase. The myelin sheaths owe their refraction to polarized light to the presence of the liquid crystals of the glycerophosphides, which are the only lipoids of the nervous mass capable of giving, in their semi-liquid state, any liquid crystals. They seem to represent in the fibers the principal solvents of water and of the other crystal lipoids which participate in the structure of this tissue. By means of such reciprocal physico-chemical action between the lipoids of the myelin sheaths, the content of these must be considered as a crystalloid liquid mixture in the sense that Lehmann describes these formations. These mixtures consist of at least five substances, among which cholesterin, water, and sphingomyelin give true crystals and not crystalloid liquids.

Anisotropia does not occur in nerve cells, and therefore mixtures containing glycerophosphide do not exist in crystal liquid

form. Myelin drops however are formed within them during pathologic processes and the metamorphosis of regression. It is not possible therefore to exclude the possibility that an abnormal acquisition of water by the phosphatids contained in them may be the reason of such formations. In the pathologic analyses already analyzed, the increase of water is a constant finding. If its action upon the phosphatids is explained in the tissue as it is *in vitro*, the successive emulsion of a colloidal nature of those lipoids and their diffusion in the aqueous media would be explained, occurring in connection with the loss of their myelinic form. Cholesterin which has not this marked affinity towards dispersing its base in water, would be held *in situ* by its lipid mixtures. I referred above to its property of markedly elevating the superficial tension of its solvents, and in order to maintain the equilibrium of their tension, which the liberation of amphophilic colloids tends to diminish, the function of the semi-colloid cholesterin must be exercised as a protector of cellular juices. One must keep in mind in valuating the reasons for the permanence of cholesterin this fact without losing sight of its other marked antitoxic properties, for example its being linked with saponin and digitoxin and also its antihemolytic power, etc.

I have summarized the physico-chemical properties of the cerebral lipoids in order to take up a possible analogy in the behavior of these processes *in vitro* and those which might be discovered *in vivo* in many degenerative pathological states. The increase of water, the lack of change in the cholesterin even though this may be detached from its proteid and lipid mixtures, and the formation of myelin drops and of phosphatid and cerebrosid mixtures in the ganglion cells and in the entire tissue which is the prey of pathologic processes, and the poverty of the tissue itself in these lipoids as compared with the normal, find in truth some means of being interpreted by means of the single physico-chemical properties of these substances and of their behavior in their mixtures.

The attempt is made through these considerations to give a biochemical interpretation of many of the pathological processes occurring in mental and nervous diseases—a temporary interpretation, which awaits new light concerning other properties of the substances under examination, and of their mixtures and the biological phenomena which accompany them.

I have put in evidence a type of lipoproteic degeneration of the nervous mass which seems to occur by preference in many disease processes; but this does not deny that degenerative processes may occur differently in the central nervous system from similar states in the peripheral. In many experimental intoxications one sometimes finds different pictures from those just described, even though these researches are limited and incomplete. Novi and Pellacani, experimenting with dogs intoxicated with both chloroform and alcohol, found in the brain an increase of water and phosphatids and a diminution of cholesterin. Sieber however in dogs mildly intoxicated with alcohol and killed while their condition was still good, found a diminution in phosphatids. Duceschi found an increase of cholesterin and of phosphatids in the blood of alcoholic animals. Bacillari also found hypercholesterinemia in individuals of alcoholic habits. With ether and chloroform narcosis there is an increasing content of cholesterin which goes hand in hand with the depth of anesthetics (Reicher, Hueck, Capporetti). It would seem therefore that narcotics provoke a chemical modification in the brain which is translated into an increase of water and a diminution of cholesterin. Concerning the phosphatids the results are not clear. It is not improbable however that the quantitative variations in these substances are in proportion to the duration and the intensity of the intoxication and in the first place when the period of intoxication is prolonged the lipoids become liberated from their lipoproteid mixtures, the cholesterin diffusing with great ease, breaking up into the aqueous emulsivity *in situ* and tend to absorb in part the circulating phosphatids. The in-

crease of water has even here a significance analogous to that discussed above. These experiences however do not yet attest to a lipoidolitic process occurring in the presence of narcotics, being much too incomplete. A more extensive research into these fields is necessary.

The scope of these brief notes is to point out in a large way the problems which present themselves. I have pointed out above some questions which concern the neurologist and psychiatrist, but for the chemist and biologist the horizon is wide open for the ambitious. Facing the study of the pathology, construction and physiology of the nervous system with all the means offered by the biological and general sciences, the biochemist proposes to arrive at the solution of these problems upon an experimental base of facts comprised within the laws which regulate the phenomena of energy and matter. Because the material is nothing but a combination of particular types of energy, the supreme scope of this method of study is to apply to the organ which presides over nervous and psychic energy the ordinary laws of energy, and to explain its phenomena upon the basis same as the latter.

We must have faith in the future of this science and its methods. The work must be methodical and organized, because many solutions of those problems which most puzzle us appear to await only intensive research. It is only necessary to explore this rich territory with the measured foresight used by the skilled artisan to calculate in advance the fruits of labor. Of these themes, above all others the chemical constitution of tissue calls us. Besides the edtte as Thudichum explains, which have been isolated, there are others which must be identified and their properties studied, whether they are minerals, extracts, lipoids or proteins. We recall how the mineral exchange of tissues is closely connected with the hormonic exchange. How many minerals have a co-enzymatic function; such as manganese, iron and the fluorine contained in the brain. Many radio-active substances, for example potassium, which is rich in the nerve cells, must

be studied; and we are reminded also of their ionic action, which is especially important in the phenomena of polarization and depolarization of the cellular elements.

The knowledge of the chemical constituents and their distribution in the various parts of the nervous system brings us to the chemical anatomy of the tissue which must be the basis of future morphology. The histochemistry of substances or groups and mixtures of substances must be differentiated in the cellular and plasmic fluids. Chemical pathology must be extended to all forms of nervous and mental disease which await new impulses obtained from normal analytical studies.

Closely linked with this question is that of the elective alteration of parts of the nervous system as regards poisons, toxins, etc., and their elective fixation, neutralization and exaltation of tixic activity. The study of the physico-chemical laws which regulate these mutual changes must be made comparatively *in vitro* and *in vivo*; and it should be extended with appropriate methods to the hormones and ferments. The close connection between endocrine secretions and the nervous system finds every day great documentary evidence through much varied research. There is lacking however a systematic study of these things, which merits the employment of a single laboratory devoted to the problems. This is true also of the fermentative processes and of the study of anomalous pathological ferments.

In connection with these analytic studies arises the problem of the structure and histologic constitution, and also of the superficial energy and tension and of the electric potential, and also the force and direction of the liquid crystalline molecule. The matter of their dielectric and electro-motor force must be considered.

In this last direction recent work gives much promise. We have seen how the anisotropia of the nerve fibers seems to depend upon the crystal liquids of the glycerophosphides in mixtures with the cholesterin and the cerebrosides. These mixtures construct the myelin sheath and give it its super-

ficial elasticity in the sense of Fresnel. The myelin sheath upon optical examination behaves as if it were composed of single axial particles with optic axes disposed radially along the transverse plane of the normal fiber. In their cylindrical space the mass remains under a superficial pressure which depends upon the cohesive force of its particles. Under the action of this pressure which is exerted radially and the force of direction which is exerted by the crystalline particles in the liquid, the one upon the other, there is originated and kept up a regular orientation of these particles, which manifests itself to polariscopic examination by the radial disposition of their optic axes. An analogous picture is seen in the structure of other crystalline bodies (quartz). This should correspond to the direction of the higher dielectric constant.

This last deduction, which has been confirmed experimentally, has great significance. Following in the fiber a higher dielectric constant the radial direction, by this means must discover by preference the chemical reaction and the chemico-physical make-up of the sheaths, the velocity of reaction being in strict correspondence to the dielectric capacity of the medium (Menschutkin). For which reason any cause capable of modifying such a dielectric capacity must also modify the velocity and therefore the function of the fiber. We have noted the connection between lipoids and organic solvents. Whether it is that they form suspensions or solutions between the two kinds of lipoids discussed, those solvents have a tendency to divide themselves abundantly between the aqueous and lipid phases. Keeping in mind that all these solvents have a low dielectric constant (alcohol 25, 9, chloroform 5, ether 4, 4, as regards water 77) and that they have narcotic powers, the hypothesis is that such substances also function as narcotics, lowering the specific conductivity of the medium, and thus the velocity of its reaction which, as regards its function, may be translated into a diminished capacity to conduct stimuli.

Between these reactions one expects to

find a high biologic significance; the development of heat and electricity by means of thermic and tactile agitation acting upon the system of liquid crystals which compose those sheaths. These crystals are symmetrically disposed and contained in certain ways above indicated by forces of cohesion, superficial pressure, molecular direction; and each thermic or mechanical modification of one of their extremities may give a difference of potential between this and the other extremity, and in this way there may develop an electro-motor force which is analogous to what is observed in solid crystals which do not have a symmetrical center. The origin of electro motive force in these may on the other hand contribute towards an understanding of the insurgence of that special energy which we call reaction to stimuli, and we may consider it as an attribute of the irritability of nerves.

I touch lightly upon these problems in order to show how chemico-energetic investigations may illuminate them. In order to gather many facts, to clear up the properties of these substances, mixtures and structures way must be made for biologic phenomena in the field of energy, for which reason the biochemist is expected to make the greatest conquests in our branch of science. It is not possible to exclude that even psychic energy may be understood some day upon the terms of thermodynamic laws.

I add a few words of practical advice: The lack of laboratories and institutes adapted to this method of study must be corrected. We have few institutions which are provided with such laboratories of medical and physical chemistry and experimental physiology or biochemistry. Private means to endow such places and students who are willing to sacrifice themselves for the poor remuneration which is offered are also lacking. Something however is being done, but laboratories must be materially endowed so that students may follow in a systematic way this experimental work, without anxiety as to expense.

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THE EVOLUTION AND FUNCTIONAL SIGNIFICANCE OF THE MEDULLA OBLONGATA*

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I

ENCEPHALIZATION AND A GENERAL VIEW OF THE MEDULLA

The prolongation of the Spinal Cord into the Head.—The portion of the neuraxis protected by the vertebral column is the *spinal cord* or *myelon*, while the portion to which the skull gives protection is the *brain* or *encephalon*. The transition from vertebral column to skull is marked topographically by the upper border of the atlas. That por-

tion of the neuraxis above this border and within the skull is the brain. In passing from vertebral column to skull there are many striking differences in form and appearance, which depend upon the development of the head. Similar differences are observed in passing from the spinal cord to the brain. These latter changes, however, are more

*From the forthcoming book, "Form and Functions of the Central Nervous System," by Frederick Tilney and Henry Alsop Riley, to be published by Paul B. Hoeber, New York.

gradual. The caudalmost portion of the brain has therefore much in common with the general appearance of the spinal cord. It is for this reason often called the "prolongation of the spinal cord," but more usually is referred to as the *medulla oblongata*. This part of the brain is also known as the *myelencephalon*, thereby denoting the region of transition in the neuraxis which presents features common to both *myelon* and *encephalon*.

The medulla oblongata, studied as a division of the human central nervous system, has proved a structure difficult to understand. Its organization seems to differ much from that of the spinal cord. Yet the fundamentals of the neuraxis seen in the spinal cord are also to be found in the medulla oblongata. They have become modified and supplemented in many ways. When, however, the reasons underlying these modifications are appreciated, the significance of the medulla is easily understood. The myelencephalon, like the myelon, is a segmented structure. But, being a segmental portion of the neuraxis, why have its segments become so profoundly altered? This question leads back into the ancient history of the vertebrates. It is involved in the record of those advances in animal life which have eventuated in the development of the head.

Influences Determining the Formation of the Head.—Long before the age of vertebrates, influences which determined the formation of the head were at work. The oldest part of the head is the mouth; its chief, primitive activity was the capture and introduction of food into the body. Even anemones and corals have well-defined mouths surrounded by sensory tentacles which aid in procuring food. The mouth, of necessity, became the point of approach which guided the animal to food supply. It thus determined the direction of locomotion and in this manner laid the cornerstone for the extensive superstructure ultimately developed at the cephalic extremity of the animal. This process which resulted in the formation of the head is known as encephalization. It received its initial impetus

through the effects of directing animal motion, and rapidly began to expand its field of influence by the accession of organs whose purpose was the more effectual guidance of locomotion.

The addition to the head of primitive visual organs, such as those seen in the flat worms, was a distinct gain in directing motion. The concentration of certain nervous elements to form primitive gustatory and olfactory apparatus as well as highly specialized tactile organs, the tentacles, brought new accretions of power to the cephalic region. The head became the chief guide of action in the lower forms, in which it foreshadowed the ultimate dominant control of the brain over the activities of the higher animals.

Influences Producing Modifications in the Head.—Food supply in the primitive race for life is never a gratuity, and no principle has been more availing to secure existence and stimulate progress than the law of might. The head, therefore, developed as an efficient instrument of attack, and was, at the same time, well provided for the defense of the delicate organs which it contained. The mouth came to be surrounded by bony jaws equipped with teeth and the head was incased in a tough skin or covered with armor-like scales for protection.

The vertebrates in their beginning showed this specialization for offense and defense in a high degree. Certain ancient fish-like animals of the Silurian and Devonian ages had a head covered by bony skin which served as a cuirass and helmet.

Among the lower vertebrates, the sharks present all of the familiar features of the head, including the mouth surrounded by jaws which have a formidable equipment of teeth, the tongue and the lips, the nostrils and the well-protected eye-balls. How much these advantages of the animal's head have added to the effectiveness of seeking and capturing its prey, as well as providing defence against its natural enemies, is obvious at a glance.

The Requirements of Respiration—The Gills.—As distance receptors, the eyes, taste-

buds and olfactory organs serve as the essential directors of motion, both in pursuit and flight. In the fish, however, the head has assumed several other responsibilities. One of these activities is connected with the mouth. Primarily serving for the intake of food, the mouth also became implicated in the function of respiration. This new function was intimately associated with the development of certain aërating organs, the *gills*. These structures consist of a set of vascular fringes situated upon either side of the head in connection with the mouth-cavity. Their arrangement permits the water to circulate freely about the vascular fringes which constitute an aërating apparatus. In this manner oxygen is taken up by the blood from the water. The gill apparatus, in addition to this provision which it makes for the respiratory necessities of the animal, presents peculiarities in form which are equally notable. Like the body segments, the gills are arranged as a series of similar structures which vary in number from five to seven and consist of gill arches with gill clefts between them. Appended to each arch is a highly vascular gill fringe. This regular series of structures in the head region, serving the purposes of respiration, is derived from the *branchiomeres* or gill segments. These segments differ in respect to their origin from the body segments, which arise from the *metameres*. In the same manner that the metameres have made a deep impress upon the spinal cord, so the segments which developed in the head region in relation to the gill arches have made a correspondingly deep imprint upon the medulla oblongata.

Encephalomeres and Myelomeres.—It is not surprising that the type of segmentation should differ in the spinal cord and the brain-stem. McClure has made a nominal distinction between these types by terming the segments of the spinal cord *myelomeres* and those of the brain-stem *encephalomeres*. The explanation of the chief differences between these two varieties of neural segments is found in the two types of segmentation which impress themselves upon the central

axis. The *metameres* (body segments) are a regular succession of equivalent segments in the region of the spinal cord. The wholly different and yet meristic series of *branchiomeres* (gill segments) is dominant in determining the form of the medulla oblongata. The major segmental influence upon the brain-stem is derived from branchiomic segmentation. The effects of metameric segmentation, although overshadowed by the gill arches, may be discerned to some extent in the medulla. Although the encephalomeres are a series of equivalent structures, they do not present any exact equivalency with the myelomeres. The encephalomere, in the medulla at least, is morphologically and physiologically different from the myelomere. It takes its chief importance from the rôle it plays in control of the gill apparatus. Its function is most intimately connected with the regulation of respiration and is, therefore, essentially *splanchnic*. The functions of the myelomeres are more especially *somatic*.

Control of the Medulla Over Respiration, Cardiac Action and Gastrointestinal Activity.—From the association of the gill mechanism with the process of aëration of the blood, it may be assumed that the chief organ of circulation would be situated in close relation with these arches. It is for this reason that in all of the fish, and in all animals depending upon gills for the aëration of the blood, the heart is situated in close proximity to the gills themselves. The distinct autonomy of the myelomere over its corresponding body segment has already been discussed. It has been seen that the principal office of such a segment of the nervous system is the control of somatic activity. The major control of the encephalomere, particularly in the region of the medulla oblongata, is concerned in the regulation of those receptors and effectors which cooperate in the process of aëration of the blood. This is a *visceral* or *splanchnic* activity. The encephalomeres of the medulla oblongata control the activity of the heart in relation to aëration of the blood and general systemic circulation. The influence of this early re-

sponsibility of the medulla in regulating respiration and controlling circulation has been transmitted through the entire line of vertebrates. It is present in those forms which came after the lungs had replaced the gills and the aquatic mode of life had yielded in favor of air breathing and other terrestrial habits.

The primitive relations of the mouth as an aperture for the intake of food and for aërating the blood have determined a close association between the cardiovascular, alimentary and respiratory systems. This relationship is reflected in the influence which the medulla oblongata has continued to exert over these three systems. The control of deglutition and gastrointestinal activities, respiration and cardiac action has come to be an autonomy vested in the medulla oblongata. From this fact the organ takes its chief importance in the regulation of life and has gained its reputation as the "*nodus vitalis*" (vital node).

The Necessary Increase of Gray Matter in the Medulla.—The requirements of the regulating of these vital processes have imposed upon the medulla certain modifications of form for which there are no analogues in the spinal cord. Because it is a portion of the nervous system which holds such autonomous control, it has, of necessity, developed more particularly along the line of its gray matter, the active substance. The result of this development is seen in the enlargement of the medulla oblongata as a whole when compared with the spinal cord. It is further evidenced by the appearance of the fourth ventricle, which has permitted the expansion of the central gray matter. The fourth ventricle is a chamber resulting from the failure of fusion of the two alar laminae. The central gray matter in this way acquires greater proportions than in the spinal cord. In some of the fish, this ventricle is not only an extensive cavity, but also presents upon its floor a series of longitudinal columns or lobes of gray matter. The most prominent of these lobes is the *lobus visceralis* which controls respiration and cardiac activity. It also has a gustatory function. The *lobus lineae*

lateralis and the *lobus somaticus* act in the direction of motion and the regulation of body balance.

The Development of the Fourth Ventricle.—The primitive central canal of the spinal cord is entirely surrounded by gray matter. In the medulla it becomes dilated to form the fourth ventricle. In the earliest phase of development, the central nervous system presents itself as a slightly differentiated neural plate. Subsequently the neural groove is formed by the appearance of the two neural folds. These folds rising gradually ultimately fuse in the mid-dorsal line, thus giving rise to the neural tube which encloses a large central cavity, the neural canal. This canal is surrounded by the central gray matter. As development proceeds the gray matter in the spinal cord is enveloped by a medullary velum consisting of neuraxones, the beginning of the white matter. In the medulla, however, there is no disposition on the part of the medullary substance to envelop the central gray matter. The extreme development of the central gray matter seems to preclude such envelopment by white substance. For this reason the central canal remains open and thus facilitates the expansion of the central gray substance.

The Development of the Gustatory Sense.—The medulla oblongata through another important function serves to increase the animal's efficiency in the search for and collection of food. This object is accomplished by the aid of the gustatory or taste sense. In many fish taste-buds are distributed throughout the mouth along the gills and head. Some of the bony fish are still more extensively equipped with these organs which are richly distributed over the fins and body.

The Development of the Equilibratory Mechanism.—In addition to this visceral regulation the medulla oblongata has assumed certain functions concerned in maintaining the balance of the body. These balancing activities, particularly in aquatic animals, are constantly in demand. The case is different with terrestrial animals, whose requirements in balancing are more transitory

and irregular. The fish, because of its continuous needs in equilibrium, requires a more extensive mechanism for this purpose. It lives in a fluid medium and must constantly maintain its body in a certain position. No such uninterrupted demand is made upon terrestrial animals. Their medium is not only more stable, but also furnishes firm support for the body.

In the fish provision for a balancing apparatus is made in two special sets of organs whose central control is vested in the medulla oblongata. The first set consists of a series of *semicircular canals* situated in the head. These canals are so arranged as to act in the manner of water levels for the three planes of space. The semicircular canals seem to regulate *dynamic equilibrium* while the animal is in motion. Ancillary portions of this apparatus, the *utricle* and *sacculus*, serve the purpose of static equilibrium while the animal is at rest.

The second set of structures in the balancing mechanism comprises the *lateral line organs* or the *neuromast system*. This system consists of a collection of specialized receptors situated upon the head in supra-orbital, infraorbital and hyomandibular rows. It is also arranged in a line along the sides of the body extending from the head to the tail and occupying a position midway between the dorsal and ventral fins.

These two sets of balancing organs provide for the static and dynamic equilibrium of the body. They furnish a balancing mechanism governing the movements of locomotion and rest.

The Development of Special Organs of Offense.—A further tendency for specialization in the medulla oblongata is seen in the electric lobe which develops in certain fish. This electric organ consists of a large collection of nerve cells connected with specialized effectors upon the surface of the body. The electric lobe serves the animal in overcoming its quarry. It generates an electric force, sufficient to stun or momentarily to paralyze its prey. Such an organ is found in one of the Selachians, the *Torpedo*.

The Primitive Functions of the Medulla.

—The primitive function of the medulla oblongata is the control of those visceral activities necessary to maintain life. The manner in which the medulla came to be the dominant regulator of respiration, cardiac action, deglutition and digestion, seems clear. All of these functions are closely related and their organs intimately connected. The proximity of the gills to the heart, of the mouth to the pharynx and stomach, bespeaks a close functional association indicative of the autonomy in the medulla which dominates the vital processes. These functions have been supplemented by others which made possible the most precise direction of body movements, and in some instances have provided a mechanism, such as the electric organ, to facilitate the capture of prey.

Modifications Due to the Assumption of Terrestrial Life. Air Breathing.—During the critical epoch when vertebrate development led to the transition from aquatic life to terrestrial habitat, a decisive change in environment produced far reaching effects upon all parts of the body. These changes have been especially reflected in the medulla oblongata. Air instead of water now served as the medium to bring oxygen to the blood and the type of respiration was profoundly altered. The mechanism of the gills, which provided for respiration in water, was no longer adequate to the demands of air breathing. After many progressive modifications the structures necessary for the development of the pulmonic system finally made their appearance. In producing these changes, what is old has been made use of in the new. The central nervous system in particular has adhered to its ancient lines. In the terrestrial animal the medulla oblongata still preserves its regulating influence over the new order of respiratory mechanism. It also maintains its dominant control of cardiac functions and gastrointestinal action.

Recession of the Taste-buds and Lateral Line Organs.—The disappearance of the taste-buds from the head, from the gill arches and from the surface of the body,

decreased the importance of the lobus visceralis which became reduced in size. Upon the assumption of terrestrial habits, the lateral line organs ceased to be of service, since the medium on which they depended no longer surrounded the body. This change eventually led to the disappearance of the lateral line system, which, however, was replaced by a new set of receptors sensitive to the stimuli of sound. By gradual transition the open *pit canal organ* of the lateral line came to be differentiated as the ear, which furnished if not a wholly new sense mechanism, at least one profoundly modified and susceptible to the stimuli of rapid air waves. The vestibular mechanism in consequence of this modification, received a new element in air breathing animals. The ear eventually assumed a dual function, i.e., the ancient one handed down from aquatic life and dependent upon the semicircular canals, the utricle and saccule; and a second function probably arising out of the highly modified lateral line system which gave rise to the cochlea and made possible the sense of hearing.

Phonation and the Development of the Larynx.—It is probable that no circumstance in the evolutionary process reveals more directly the results of cause and effect than the development of phonation in response to the appearance of the organ of hearing. The auditory sense provided the animal with new information regarding its environment. It also made possible a means of communication which much increased the range of herd activities and ultimately enriched racial experience by the addition of one of the most effective instruments in social relations, the voice. Without the sense of hearing, the voice would have no reason for being. This is well shown in those born deaf, for in deaf mutes, the development of the voice is either negligible or abnormal.

At the time when the lungs made their first appearance, they developed as an off-shot from the gastrointestinal canal. Because of this relation, food and air have a common passageway for some distance from

the mouth. At a certain point, however, a critical selection became necessary whereby the food should follow its own course and the air enter passages especially designed for it. There could be no intermingling of these two elements, the food and the air, at this critical point. In response to this need, a sensitive mechanism composed of a set of delicate valves was established at the junction of the food and air canals.

The lungs especially required protection against the entrance of food, and out of this need arose the glottis, the epiglottis, and the larynx with its highly specialized vocal cords. The intimate relation between the lungs and the gastrointestinal tract accounts for the control which the medulla has over both systems. It is but a natural consequence that this part of the brain should also regulate the mechanism which has guarded the entry into the lung and eventually developed into the organ of voice.

The Development of the Mobile Head and Tongue.—Another decisive change came with the appearance of the neck and the development of the mobile head. This modification was determined in the interest of greater range in directing movements of the body and extremities. The increased mobility complicated the function of balance. It introduced new planes of motion between the head and the trunk and created new demands upon the balancing mechanism. It also added much to the function of equilibrium as concerned in the movements of the eyes and the head together.

The addition of the mobile, muscular tongue implicated the medulla in another function and complicated its branchiomeric control by the inclusion of the myotomes of two or three body segments (lingual metameres). In this light the segmentation of the medulla, at least in the higher vertebrates, must be complex. It is fundamentally influenced by the gill segments, but is later supplemented by the addition of at least two or three metameric segments. The encephalomere of the medulla is not, therefore, the equivalent of the myelomere. In the higher vertebrates it represents a branchial segmen-

tation to which is added an element of metameric segmentation.

Modifications of the Face.—Certain changes in the face also made their impress upon the medulla oblongata and determined the development of one of its most prominent nuclei. The face in the lower terrestrial vertebrates consists of a bony mask covered by skin, quite incapable of facial movements and devoid of facial expression. When the scaly skin is removed from the facial portion of the skull in the reptile, no facial muscles are found. This immobile and non-muscular face also occurs in birds. It is only in the mammals that the soft muscular lips and the muscular layers about the nose, eyes, ears and forehead make their appearance. Equipped with this facial musculature capable of expressing various emotions, the mammal has acquired a new instrument for adjustment to its social environment. The expression of anger serves as a warning against encroachment, as that of hatred is a warning against impending attack. These and many other emotive expressions furnish an effective equipment for social adjustments possessed only by such animals as have a muscular, mobile face.

The origin of the facial muscles is a matter of dispute. It seems probable that the facial nerve supplying these muscles had its inception in the nerve fibers which originally supplied the platysma muscles on the surface of the throat, as well as the immediately underlying sphincter colli muscle. This sheaf of subcutaneous muscle has gradually

shifted upward to take its place over the old bony mask of the vertebrate head beneath the skin. It has carried with it the original innervation through the facial nerve. It seems probable that these primitive muscles, situated in the region of the neck and ultimately spreading to the face, arose as part of the gill apparatus. They would in this sense be derivatives of the gill muscles which were supplied by the post-trematic branch of the seventh nerve in the fish.

The Influence of Suprasegmental Structures.—Among the other influences modifying the form of the medulla must be mentioned the greater need for equilibratory control witnessed in the development of the cerebellum and the introduction of volitional control attending the appearance and expansion of the cerebral hemispheres.

The medulla oblongata, from the beginning of vertebrate organization, has comprised a series of segments in which was vested the autonomous control of cardiovascular, respiratory and gastro-intestinal activities. To these offices have been added the regulation of body movement in the interests of equilibrium and a special mechanism which made the auditory function possible.

The interpretation of the anatomy of the medulla will be aided by holding these facts in the foreground. The explanation of the modifications to which this organ has been subject in man must be sought in the progressive adaptations developed in consequence of special adjustments to terrestrial life.

II

THE FUNCTIONAL SIGNIFICANCE OF THE MEDULLA OBLONGATA

Functions of the Gray Matter.—The functions of the gray matter of the medulla oblongata may be classified with reference to the components through which they act. The chief function of this portion of the brain is represented by the splanchnic autonomy which the medulla holds over the vital processes of respiration, cardiac action, deglutition and intestinal activity. This

autonomy is expressed in the activities of the splanchnic motor and splanchnic sensory components of the nervous system.

In addition to these splanchnic activities, the gray matter of the medulla acts as a relay station for several qualities of somatic sensibility and mediates influences which control the action of certain somatic muscles.

Splanchnic Motor Functions of the Medulla.—Through the dorsal motor nucleus of the vagus and glossopharyngeus nerves, the medulla innervates many glandular effectors and involuntary muscles. Among these motor and glandular effectors are:

1. The muscles of the soft palate.
2. The constrictors of the pharynx.
3. The muscles of the esophagus.
4. The muscles of the stomach.
5. The muscles of the intestines and part of the colon as far as the descending colon.
6. The musculature of the bronchial tree and trachea.
7. The musculature of the heart.

It also innervates the salivary glands through the inferior salivary nucleus as well as exerting an effector influence over the glycogenic activity of the liver and the secretions of the pancreas. Through the vagus and the medulla aids in activating the glands of the stomach and intestines.

The innervation of the fauces and pharynx is furnished by the glossopharyngeal nerve, while the lower portions of the respiratory and gut tracts are regulated by the vagus. The medulla oblongata controls the muscular activities of the larynx, the impulses to which are supplied by the nucleus ambiguus. It also in part controls the facial muscles, through the motor nucleus of the facial nerve which is the final common pathway for the musculature of expression and the platysma myoides muscle.

Splanchnic Sensory Functions of the Medulla.—Through the sensory nuclei of the vagus and glossopharyngeus nerves, the medulla affords the following areas their splanchnic sensory supply:

1. Part of the dura mater in the region of the sinus transversus.
2. The soft palate.
3. The posterior third of the special sense of taste; in this region it also supplies the mucosa of the Eustachian tube and through Jacobson's branch of the glossopharyngeal nerve, the tympanic cavity, the fenestra ovalis, the fenestra rotunda and the mucosa of the mastoid cells.
4. The pharynx and epiglottis.

5. The larynx.
6. The trachea.
7. The respiratory mucosa.
8. The mucosa of the esophagus.
9. The mucosa of the stomach.
10. The mucosa of the duodenum, jejunum and ileum.
11. The mucosa of the bile ducts.
12. The mucosa of the ascending and transverse colon.
13. The heart and pericardium.

Concerning the supply of the sense of taste to the posterior third of the tongue, the arch of the palate and the uvula, there is some difference of opinion. According to certain authorities, the trigeminal nerve supplies the sensory element and the ninth nerve the special taste innervation. According to others, the glossopharyngeal nerve is wholly sensory, while the trigeminal is the taste nerve; some believe that each of these two nerves has a dual function, in part sensory and in part taste. The central receiving station for the sense of taste is the nucleus fasciculus solitarius. The root fibers entering into connection with this nucleus arise chiefly in the seventh and ninth nerves, although the tenth nerve may make a small contribution to it.

The medulla innervates the anterior two-thirds of the tongue with special splanchnic sensory fibers of taste through the *chorda tympani*, the sensory division of the facial nerve.

Somatic Motor Functions of the Medulla Oblongata.—Through the hypoglossal nerve the medulla supplies all of the intrinsic muscles of the tongue. These muscles are derived from myotomes and hence the tongue is to be regarded as belonging to the somatic motor system.

By means of the inferior olive, the medulla furnishes an important center for the coordinative control of the eye and head movements together.

The functional significance of the inferior olivary body is not clear at present. Bechterew regards it as a center for static equilibrium. This supposition does not seem to accord well with the facts. The olive reaches

its highest development in those animals requiring the most exact cooperative coordination between eye movements and movements of the head. The anatomical connections of the olive lead to the belief that its function is the coordination of these movements. Its connection with the cerebellum through the olivo-cerebellar fibers, with the nuclei of the oculomotor apparatus through the central tegmental tract, and with the upper cervical segments of the spinal cord through the tract of Helweg, seem to point to a mechanism which is especially well adapted to bring into relation movements of the eyes and of the head.

The Somatic Sensory Functions of the Medulla Oblongata.—The medulla oblongata serves as the chief relay station in the pathway of impulses arising in the inner ear and proceeding to the auditory area of the brain to serve the purposes of audition. These fibers develop in connection with the cochlear portion of the eighth nerve. The more primitive division of the eighth nerve, that which is connected with the semicircular canals, the utricle and sacculi, also receives a relay in the medulla. This relay connects with certain areas of the brain involved in equilibratory control. According to some authorities, the fibers arising in the organs of the vestibular system belong to the proprioceptive system, as do also the fibers which arise in the muscles, joints and bones. There can be no objection to considering the vestibular fibers as integral parts of the proprioceptive system, provided it is recognized that their end organs are genetically related to the ectoderm. The end-organs of the muscles, joints and bones, on the other hand, are related to the mesoderm. The medulla, by means of the nucleus cuneatus, the nucleus gracilis, and the substantia gelatinosa, affords relays for the discriminative types of sensibility coming from the leg and lower trunk, from the arm and upper trunk, from the back of the head and face. It is probable that through the relay of the substantia gelatinosa all types of sensory impulses are transmitted to the cerebral cortex.

A small aberrant branch of the vagus nerve supplies a cutaneous area in the external ear immediately surrounding the external auditory canal. This innervation is effected through Arnold's nerve.

Simple Reflexes Mediated Through the Medulla Oblongata. The coughing reflex.—This reflex may be determined by stimulation of the mucous membrane of the pharynx, of the larynx, of the trachea, of the bronchial tree or the external auditory canal. The afferent impulses are conveyed by branches of the vagus nerve. They reach the dorsal nucleus of the vagus and are transmitted to the respiratory nucleus of the medulla which occasions a forceful expiratory effort preceded by a deep inspiration.

The swallowing reflex.—This reflex is occasioned by mechanical stimulation of the wall of the pharynx and is activated through branches of the vagus and glossopharyngeal nerves. The motor center of the reflex is in the dorsal vago-glossopharyngeal nucleus. Section of the glossopharyngeal nerve causes the disappearance of the swallowing reflex.

Vomiting or regurgitation reflex.—This reflex may be determined by abnormal stimuli in the region of the cardia of the stomach, in the wall of the pharynx, in the gastrointestinal tract, or in the internal ear. The afferent arm of the reflex is furnished by the vagus and glossopharyngeal nerves. The motor center is in the dorsal motor nucleus of the vagus, a special subdivision of which is sometimes spoken of as the *vomiting center*. The efferent arm of the reflex is furnished by the vagus nerve.

The salivary reflex.—This reflex may be determined by stimulation of the mucous membrane of the vestibule of the mouth, of the lips or of the general mucosa of the oral cavity by gustatory stimuli such as chemical or mechanical stimuli. Stimulation of the chorda tympani, of the oral distribution of the trigeminal or of the glosso-pharyngeal area, will cause the passage of afferent impulses to the nucleus salivatorius inferior in the medulla and determine effector impulses activating the salivary glands.

The sneezing reflex.—This reflex may be determined by afferent impulses received from the mucous membrane of the nasal cavity by way of the trigeminal nerve. When transmitted to the respiratory nucleus in the medulla they activate a forceful expiratory effort, forcing the air through the nasal passages under considerable pressure.

The sucking reflex of the new-born may be regarded as a simple reflex, inasmuch as it is determined by the presence of an object in the mouth of an infant, thus stimulating the trigeminal and glossopharyngeal nerves to transmit sensory impulses to the medulla from which arise motor impulses distributed through the hypoglossal, glossopharyngeal and facial nerves, bringing the respiratory centers into play at the same time. In later life this reflex is conditioned by control from higher divisions of the brain.

THE MEDULLA IN ITS RELATION TO SPECIAL FUNCTIONS

The medulla oblongata has autonomous control over certain vital functions, chief among them being those of respiration, cardiac activity, ingestion, deglutition and digestion of food, circulation, metabolism and secretion.

The Medulla Oblongata in its Relation to Respiration.—In considering the relation of the medulla oblongata to the function of respiration, it is not proposed to discuss the physiology of this mechanism as a whole, but rather the rôle which the myelencephalon plays in it.

The muscle groups active in the respiratory mechanism.—The respiratory mechanism brings into action certain muscular groups of the body. These may be classified as (1) the principal groups; (2) the ordinary accessory groups, and (3) the extraordinary accessory groups. In extreme emergency, it is probable that few muscles of the body are exempt from participation in the respiratory effort.

1. *The principal muscle groups active in the respiratory system:*

a. The chief muscles acting in respira-

tion are the diaphragms, which are supplied by the phrenic nerve, the latter arising in the third, fourth and fifth cervical segments of the spinal cord.

- b. The external intercostal muscles and the intercartilaginous muscles, which are supplied by the intercostal nerves arising in the thoracic segments of the spinal cord, from the first to the twelfth inclusive.
- c. The long and short levator of the muscles of the ribs supplied by the dorsal branches of the intercostal nerves.

These groups of muscles act in ordinary inspiration, while in ordinary expiration there is no actual muscular contraction, since the elasticity of the tissues of the thoracic basket, together with that of the lungs and the abdominal muscles, determines the expiratory movement.

2. *The ordinary accessory muscles active in the respiratory mechanism:*

- a. The scaleni muscles, innervated by the cervical and brachial plexus and arising in the upper cervical segments of the spinal cord.
- b. The serratus posticus superior muscle, supplied by the dorsalis scapulae nerve which arises in the cervical segments of the spinal cord.
- c. The sterno-cleido-mastoid muscle, supplied by the spinal accessory nerve.
- d. The trapezius muscle, supplied by the spinal accessory nerve.
- e. The rhomboid muscles, supplied by the dorsalis scapulae nerve, which arises in the cervical segments of the spinal cord.
- f. The pectoralis minor muscle, supplied by the anterior thoracic nerves.

Certain muscle groups serve to assist the respiratory act by increasing the capacity of the passages admitting the air. These are:

- a. The depressors of the larynx, the sternohyoideus and the sternothyroideus muscles, supplied by the descendens hypoglossi nerve.

- b. The crico-arytenoideus posterior and the thyro-arytenoideus separate the vocal cords. These muscles are supplied by the recurrent laryngeal nerve, a branch of the vagus.
- c. The depressors of the lower jaw supplied by the trigeminal, facial and hypoglossal nerves.
- d. The depressors of the tongue supplied by the hypoglossal nerve and the pharyngeal plexus.
- e. The levator palati supplied by the pharyngeal plexus.
- f. The azygos uvulae supplied by the pharyngeal plexus.
- g. The dilator narium anterior and posterior supplied by the facial nerve.
- h. The levator alae nasi supplied by the facial nerve.

In deep expiration the abdominal muscles take part by compressing the abdomen. These muscles are supplied by the abdominal branches of the eighth and twelfth intercostal nerves inclusive.

The internal intercostal muscles also act in deep inspiration to increase the capacity of the thoracic cavity.

3. *The extraordinary accessory groups active in respiration.*—In labored and extreme respiratory effort, the entire musculature of the body may act. This is seen in cases of suffocation when the whole body is thrown into convulsive movement in the struggle to gain air supply.

Respiratory Coordinating Center of the Medulla Oblongata.—The fact that such a wide range of the muscles may come into action to meet the requirements of respiration indicates the necessity of some coordinating center which shall have control over the entire skeletal musculature of the body and which, under extreme circumstances, may make use of these muscles in a most effective manner. The presence of such a coordinating center for respiration has long been suspected and is still a subject of debate.

The first exact localization of it was made by Legallois and Flourens who placed this center in the medulla oblongata in the region

of the *calamus scriptorius*. Florenz called this center the *Noeud Vital*. He believed that the center was situated deep in the reticular formation upon either side of the raphé at the lower angle of the fourth ventricle. Schiff and Girard placed the respiratory center in the *ala cinerea*, while Mislavski located it in a nucleus ventral to the hypoglossal nucleus in the reticular formation. Gad and Marinesco confirmed this general location, while Bechterew placed it in the formatio reticularis grisea on either side of the emerging fibers of the twelfth nerve. There is a general consensus of opinion that this respiratory nucleus is situated in the medulla oblongata and in the region of the gray portion of the reticular formation. According to the usual interpretation of the respiratory center, all impulses influencing the principal and accessory muscular mechanisms of respiration are ultimately delivered through this coordinating nucleus of the medulla oblongata. The nucleus not only comes under the immediate influence of reflex activity, but is controlled from the cerebral cortex by voluntary impulses. By its connection with the sensory elements of the cranial nerves, it responds to stimuli received by the receptors in the mouth, pharynx, larynx and lung, as well as in the nasal passages. It thus furnishes a center which serves to protect the air passages against invasion of deleterious substances.

Chief Source of Stimulation for the Respiratory Center.—The venosity of the blood acts as the chief source of stimulation for the respiration center of the medulla oblongata. The greater the venosity of the blood, the greater is the degree of stimulation carried to the nucleus in the formatio reticularis grisea. In addition to this stimulation, certain products of metabolism appear to activate this center, as witnessed in yawning when the individual is fatigued.

Other stimuli may affect the respiratory center. This is particularly true of olfactory stimuli received from the first cranial nerve, which cause a distinct reflex respiratory act.

Cutaneous stimuli upon the surface of the

face and head determine a reflex expiratory act, followed by deep inspiration.

Stimulation in the sensory area of the glossopharyngeus and vagus nerves is followed by a respiratory reflex movement.

Stimuli from the surface of the body, particularly a sudden application of cold or stimuli causing pain, result in a deep inspiratory act, followed by expiration.

Auditory stimuli may cause an inhibition of respiratory movement of all types which probably is in the interest of assisting the sense of hearing.

Other respiratory acts than those immediately concerned in respiration.—A respiratory act may be so modified as to give rise to phenomena quite different from those of ordinary respiration. Many of these modifications of respiration are simple reflexes, but some are determined by more complex reflex associations. Among the modifications of the respiratory act are coughing and sneezing, which have already been referred to as simple reflexes for the purposes of defense of the respiratory tract. Snuffling and other reflex respiratory activities probably belong in the same group. Snoring is a modification of breathing determined by the fact that in sleep certain changes occur in the upper respiratory passages. The respiratory act may be so modified as to give rise to yawning and sighing, both of which are indicative of changes in metabolism consequent upon fatigue and produce an increased respiratory effort in order to fill the lung with more than its usual amount of air. Both of these reactions may be regarded as the result of direct stimulation of the respiratory center, and while they are to some extent subject to the influence of the will, they are reflexes in their essential nature. Hiccoughing is another type of modified respiration, occasioned by irritations in the course of the vagus nerve. Here the usual site of irritation is in the gastrointestinal tract, but the irritant may be located in any part of the vagal distribution. Laughing and crying are modifications of respiration, resulting from excitation of the higher cen-

ters, particularly those which have to do with the regulation of the emotions.

One type of modified breathing usually regarded as pathological is the *Cheyne-Stokes* respiration. This consists of a regular series of respiratory efforts in crescendo, beginning slowly and regularly with deep inspiration and expiration, increasing in rate and rhythm and becoming more and more shallow until an actual period of apnea is reached. This period in which respiration is suspended lasts for several seconds and then the same series of deep regular respiratory movements, at first slow but passing into crescendo, appear. The *Cheyne-Stokes* respiration has been explained as due to a vascular spasm so affecting the respiratory center as to cause exhaustion of it. It should not, however, be regarded as a necessarily pathological clinical phenomenon because the same type of respiration often occurs in ordinary sleep. Although this is a common symptom of brain disease, it is not due to any known focal lesion.

The Significance of the Vagus Nerve in Respiration.—The vagus, which innervates the pulmonary tract with its sensory and motor nerve fibers, acts as a regulator of respiration. It regulates the oxygen intake to the needs of economy. This may be more graphically expressed in the ratio that the frequency and depth of respiration vary directly with the character and intensity of the stimulus; for example, during sleep the character and intensity of the stimulus is minimum and the frequency and depth of respiration is correspondingly low. In sleep there is low metabolism and less oxygen is needed, hence there is a less frequent and less deep respiratory movement. In pneumonia, on the other hand, when there is an actual air hunger because of the compromised air space in the lung, the body needs more oxygen and there is a corresponding increase in the frequency and depth of respiration. Another example illustrating this relation of the vagus to respiration is seen in the experimental animal in which both vagi are cut. In the bilaterally vagotomized dog, because

the respiratory center is no longer receiving impressions from the lungs, the normal variation in the ratio between the frequency and depth of respiration to the character and intensity of the stimulus is lost. When the vagotomized dog is placed in a chamber in which the air is gradually heated, its respiratory activity does not show the same proportional change as it does in the normal dog. In the normal animal, panting becomes conspicuous with the rise of temperature, undoubtedly due to the fact that the increased amount of heat conveyed in the air serves as a stimulus which causes respiration to deepen and become more frequent. The vagotomized dog has no means of conveying this change of atmospheric heat to his respiratory center, hence there is no proportionate variation in his respiratory movement. The vagus in its peripheral pulmonic distribution is largely a receptor responding to mechanical and chemical stimuli, which it transmits first to its primary end stations, and then to the nucleus respiratorius. The influence from this nucleus may spread to the entire musculature of the body should the needs of respiration make such demands.

The condition of the blood in the alveolar capillaries may also be active in this reflex. The vagus through the superior laryngeal nerve inhibits respiration as a defence against foreign bodies or determines modifications of respiration such as coughing in the interests of the same object. The vagus, therefore, in its relation to the respiratory cycle is a regulator, having especial control over the ratio between the intensity and character of the stimulus on the one hand and the frequency and depth of the respiratory movement on the other. The influence of the will upon respiration is not complete. It is impossible to suppress voluntarily the respiratory movement or much to increase its rate by volition. Nevertheless, most of the principal respiratory movements, together with the modifications of respiration, may be imitated at will. Sighing and yawning are slightly more subject to the will than sneezing or coughing, which are difficult to control by volition. Laughing and crying,

while essentially reflexes of the emotive sphere, are in part subject to the will. The psychic control of respiration, therefore, is but partial, and always is superseded by the reflex requirements of economy in its need for oxygen.

The Medulla in its Relation to Phonation.—Phonation, the production of the voice, and its highly modified development, speech, depend upon three primary mechanical factors which are:

1. Increase in the tension of the air in the lung behind the larynx.
2. The narrowing of the rima glottis by the movements of the vocal cords.
3. Changes in the pharynx and the mouth cavity as well as in the position of the soft palate, jaws, tongue and lips.

The muscles which determine these acts are:

1. The expiratory and inspiratory muscles.
2. The laryngeal muscles.
3. The pharyngeal, palatal, lingual, facial and mandibular muscles.

Special Functions of the Medulla in the Control of the Act of Phonation.—The medulla oblongata takes its importance in connection with this mechanism from its control of the larynx, the pharynx and part of the soft palate. In controlling the larynx, it regulates the activity of the following muscles:

1. The adductors of the vocal cords.
 - a. The thyro-arytenoideus externus.
 - b. The thyro-arytenoideus internus.
 - c. The thyro-aryepiglotticus.
 - d. The arytenoideus cisticus.
 - e. The crico-arytenoideus lateralis.
2. The abductors of the vocal cords.
 - a. The crico-arytenoideus.
 - b. The arytenoideus posticus.
3. The tensors of the vocal cords.
 - a. The cricothyroideus.
 - b. The geniohyoideus.

The medulla likewise has control of the muscular groups which serve to increase the pressure in the lungs ready for expulsion through the rima glottis. This is accomplished through the respiratory center. The larynx is supplied by the recurrent laryngeal

nerve which innervates all of the muscles except the cricothyroid, the latter being supplied by the superior laryngeal nerve. The medulla, furthermore, innervates the pharynx, the palate, the tongue and, in part, the lips, in the acts performed by these structures in phonation. A center for phonation is in all probability located in the nucleus ambiguus in the medulla oblongata.

The Medulla Oblongata in its Relation to Deglutition and Digestion.—Deglutition is a complex act having the following phases or series of movements:

The first series of movements results in grasping the bolus by the mouth which depends upon movements of the jaws, of the tongue, of the lips and of the cheeks. The movements of the jaws are controlled by the motor division of the fifth cranial nerve whose nucleus is situated in the pons varolii. The movements of the tongue are controlled by the hypoglossal nerve whose nucleus is in the medulla. The movements of the lips and cheeks are controlled by the seventh nerve whose nucleus is in part in the medulla, but mainly in the pons.

The second series of movements in deglutition result in the introduction of the bolus into the fauces. This is determined by combined movements of all of the muscle groups acting in the first phase, participating to obliterate the mouth cavity. By pressure of the tongue upon the hard palate, closure of the jaw and compression of the lips and cheeks, the bolus is forced into the isthmus of the fauces.

The third series of movements results in the closure of the posterior nares to prevent the regurgitation of food or fluid through the nose. This is accomplished by the elevation of the soft palate, thus acting as a valve to close off the orifice of the posterior nares. These muscles acting in this capacity are supplied by the ninth and tenth cranial nerves and have their nuclei in the medulla oblongata.

The fourth phase of deglutition depends upon the closure and elevation of the larynx with the object of preventing the entrance of food or fluid into the air passages

and also of opening wider the upper orifice of the gastrointestinal canal. This act is largely accomplished through the action of the myelohyoid and digastric muscles, which cause the larynx to be drawn forward and upward, while the closure of the larynx is accomplished by the epiglottis and the partial approximation of the vocal cords. The muscles causing the elevation and forward movement of the larynx are supplied by the fifth and seventh cranial nerves, part of the motor nucleus of the latter being situated in the medulla. The vagus innervates the muscles producing the valve-like changes in the epiglottis and the vocal cords, and has its center of control entirely in the medulla.

The grasp of the bolus by the pharynx takes place as a result of a contraction of the constrictor muscles of the pharynx. These muscles are innervated by the glossopharyngeal nerve which has its origin in the medulla.

Deglutition is completed by the longitudinal and circular muscle layers of the esophagus which are innervated by the vagus.

The control of deglutition is vested in the medulla oblongata with the exception of such movements as are necessary for the closure of the jaw, the occlusion of the lips and the elevation and forward traction of the larynx. These elements of deglutition are innervated by the seventh and fifth cranial nerves and thus have their main centers in the next higher division of the brainstem, the pons varolii. The greater part of the act of deglutition is reflex. Its first phase is completely under the control of the will, both in its initiation and its completion. Once the bolus of food or the fluid has passed the isthmus of the fauces, the act of deglutition becomes reflex and free from regulation by the will.

The Muscular Activities of Digestion.—The muscular movements necessary for the digestion of food are dependent upon influences derived from the medulla. These influences reach the musculature in the walls of the stomach and intestines by means of the vagus nerve. The stomach and intestines

are also supplied with motor impulses from the sympathetic system and this dual innervation acts in a mutualistic synergy, producing the movements of the gut tract most effectual in preparing the food for the chemical processes of digestion and assimilation. In the muscular act of digestion, the stomach presents three distinct divisions, each acting in a somewhat different manner.

The first portion is the *cardia*, which serves as a receptacle and as a valve preventing the regurgitation of the food into the upper food passages. Its muscular activity is in part peristaltic and in part constrictive, since it must give resistance to the active tritulatory movements which produce the necessary divisions of the food stuffs.

The second portion is the *fundus*, in which the action is rotato-tritulatory and carried on as a constant and regular peristalsis, especially during the presence of food in the stomach.

The third portion is the *pylorus*, in which the muscular activity is rhythmically peristaltic at more or less regular intervals, its object being to further the emptying of the stomach into the duodenum. It also guards the portal of the intestinal canal in such a way as to prevent the passage of food in the process of digestion until it is properly prepared for the action of the small intestine. At regular intervals the gastric contents accumulated by means of rhythmical peristalsis are propelled into the duodenum ready for further treatment in the intestinal canal.

All three parts of the stomach, acting in these different capacities, receive a dual innervation through the vagus and the sympathetic system. For the *cardia*, the vagus contains fibers which serve both as inhibitors and accelerators. These axones have their origin in the dorsal motor nucleus of the vagus. The sympathetic system is a synergist of the vagus in all of these activities of the *cardia*. The innervation of the *fundus* and the *pylorus* corresponds in all respects to that of the *cardia*, so that the stomach receives part of its dual innervation through the vagus nerve from the dorsal motor nucleus of that nerve situated in the medulla.

The muscular activity of the intestines depends upon the circular and longitudinal musculature of the intestinal canal, which is innervated in a manner similar to that of the stomach, except that in this case, the vagus acts much more as an excitator to peristalsis for the maintenance of muscle tone, while the sympathetic, according to most authorities, acts as an inhibitor.

The Medulla Oblongata in its Relation to Cardiac Action.—The heart is innervated by a triple nerve supply; first, an intrinsic innervation from the ganglia of the heart; second, through the sympathetic system; and third, through the vagus. The vagus acts as an inhibitor of cardiac action. Stimulation of the peripheral ends of the cut vagi slows the heart, and if the stimulation be strong enough will bring it to a standstill in diastole. The center for this inhibitory control of the cardiac cycle is situated in the dorsal motor nucleus of the vagus nerve. The medulla oblongata, although it is not the sole regulator of heart action, plays an important rôle in the synergizing of systole and in sparing the heart from the fatigue which would naturally ensue were it left free to pulsate under the influence of its intrinsic ganglia and the sympathetic system. Without this inhibitory control of the vagus, cardiac activity would present the phenomenon known as "runaway heart."

The Medulla Oblongata in its Relation to Metabolism, Especially to Glycometabolic Control.—It has long been known that glycosuria may be produced by a small puncture of the floor of the fourth ventricle between the nuclei of the eighth and tenth nerves. From this fact it has been argued that there is a glycometabolic center situated in the medulla oblongata. Injury of this part of the brain sometimes results in a pronounced glycosuria. This pathological condition is probably due to irritation of the vagus nucleus controlling the glandular effector fibers which innervate the liver and the pancreas. Such stimulation might easily lead to an excessive mobilization of glycogen with a corresponding hyperglycemia and glycosuria.

The Medulla Oblongata in its Relation to Secretion.—Experimental evidence has demonstrated that the medulla has control over salivary, gastric, pancreatic, intestinal, biliary, lachrymal and sudorific glandular activity. By means of fibers arising in the nucleus salivatorius inferior, the medulla exerts an influence upon the secretion of saliva. The salivary glands receive impulses from three sources; first, the inferior salivary nucleus whose fibers are furnished by the vagus nerve; second, from the superior salivary nucleus whose fibers arise in the fifth trigeminal nerve; and third, from the sympathetic system. Glandular effector fibers arising in the medulla and making their way into the stomach by means of the vagus nerve cause an increase in the secretion of gastric juice, both as a result of reflex and psychic stimuli. The vague exerts a strong stimulating influence over the pancreatic and biliary secretions which is independent of blood pressure changes. The center for the control of this secretory activity is in the dorsal vagus nucleus. The secretion of mucus throughout the gastrointestinal tract is dependent upon a dual innervation, in part derived from the vagus and also coming from the sympathetic system.

Sudoriferous secretion for the head is carried on under the influence of a center situated in the reticular formation beneath the floor of the fourth ventricle. The more exact localization of this center has yet to be determined, but clinical material abundantly justifies the idea that such a center exists in the tegmental portion of the medulla. The fibers mediating the influences which control the sweat glands of the head probably make their way to the glands by means of the facial and trigeminal nerves.

THE FUNCTIONS OF THE WHITE MATTER IN THE MEDULLA OBLONGATA

The medulla affords continuity in the conduction paths whose integral tracts arise lower down and pass upward or pass downward from origins higher up.

Descending Tracts Traversing the Me-

dulla.—As the *pyramids*, the pallio-spinal pathway for volitional control passes through the medulla without interruption. This tract is the upper neuron element in the pathway leading from the cortex to the spinal cord and bulb and thence to the skeletal muscles. It mediates influences of volitional control.

The *tecto-spinal tract*, which arises in the tectum of the midbrain, also descends through the medulla oblongata, in the more cephalic portion of which it is incorporated with the fasciculus longitudinalis posterior. In the caudal extremity of the medulla, the tecto-spinal tract moves into a more ventral position from which it passes into its usual spinal cord relations in the ventral white column. This tract transmits impulses which arise in the suprasegmental portion of the midbrain, the tectum, to the spinal cord and thus to the muscles. It is presumed that it is connected in some way with the transmission of impulses related to the perception of light and is probably concerned with the production of defence mechanisms for the protection of the eye and face by means of the upper extremity.

The *rubro-spinal tract* is an intersegmental connection which passes through the medulla without crossing and serves for the transmission of impulses which have to do with coordinative and associative automatic control.

The *Deitero-spinal tract* is another intersegmental tract connecting the nucleus of Deiter with the various levels of the spinal cord and transmitting impulses which have to do with equilibratory control. The two Deitero-spinal tracts pass through the medulla, the ventral tract partially crossing in it, while the lateral tract remains uncrossed throughout its entire extent.

The *olivo-spinal tract* likewise represents an intersegmental connection which brings the inferior olive into relation with the upper cervical segments of the spinal cord and coordinates the movements of the eyes and head.

The *central tegmental tract* is an intersegmental connection which associates the

nuclei of the third, fourth and sixth cranial nerves with the inferior olives, and thus acts as a connecting link in the mechanism which determines coordinative control of the head and eye movements together.

The fasciculus longitudinalis dorsalis of Schutz is a long intersegmental tract which has its origin in the midbrain. It represents the vestige of an old motor pathway in the neuraxis.

The reticular formation, especially the white portion, is an important descending association system which connects the respiratory nucleus of the medulla oblongata with the motor cells in the cervical and thoracic segments of the spinal cord, and serves to bring the respiratory muscles under the direct control of the principal coordinating respiratory center in the brain.

Ascending Tracts Traversing the Medulla Oblongata.—Through the medulla passes the *mesial fillet* (mesial lemniscus) which is the continuation of the columns of Goll and Burdach. In addition to the fibers of the mesial fillet, there are ascending axones from the substantia gelatinosa. These are ascending trigeminal fibers which pass through the medulla as the second neuron in the sensory pathway from the head and face.

The *spino-thalamic tract* passes through the medulla without interruption or crossing; it has already undergone decussation in the spinal cord. This tract is sometimes spoken of as the *spinal fillet* in contradistinction to the mesial fillet, which is referred to as the *bulbar fillet*. The spino-thalamic tract serves for the conveyance of pain and temperature from the opposite side of the body.

Another group of ascending fibers which pass through the medulla are the *dorsal* and *ventral spino-cerebellar tracts*. These are elements in the pathway from the muscles to the cerebellum. The dorsal spino-cerebellar tract of Flechsig becomes incorporated in the inferior cerebellar peduncle. The ventral spino-cerebellar tract of Gower does not enter into this peduncle but passes directly to the next higher segment of the brain. These two tracts are suprasegmental because they

bring the segmented portion of the nervous system into connection with a suprasegmental structure, the cerebellum.

The *posterior longitudinal fasciculus* serves to bring into coordinative relation the several nuclei in the mechanism of ocular movement. It also brings these nuclei into relation with the vestibular division of the eighth nerve, and thus under the influence of the semicircular canals.

The Principal Decussations in the Medulla.—Six pathways undergo decussation in the medulla:

1. The pallio-spinal pathway for volitional control.
2. The spino-thalamo-pallial pathway for discriminative sensibility. This is the decussation of the bulbar or mesial fillet.
3. The olivo-cerebellar pathway. This decussation is the inferior olivary crossing.
4. The spino-cerebellar pathway, which undergoes a partial crossing by means of the ventral and dorsal external arcuate fibers.
5. The trigeminal pathway, the decussation of which provides a complete crossing for all types of sensibility from the regions of the head, face and cavities of the head.
6. The Deiteral pathway, the decussation of which consists of a partial crossing of the fibers arising in Deiter's nucleus and entering either the fasciculus longitudinalis posterior or the ventral Deitero-spinal tract.

SUMMARY OF THE FUNCTIONS OF THE MEDULLA OBLONGATA

The functions of the medulla oblongata may be briefly summarized as follows:

The gray matter of the medulla embodies a dominant autonomy over the vital processes of life. It mediates an essential control over respiration, cardio-vascular activity, phonation, articulation, deglutition, digestion, secretion and metabolism. It also acts as an important relay station for the auditory nerve.

The white matter of the medulla represents the continuity in all of the major conduction paths which serve to maintain

efficient relations between the receptors and effectors of the body. The medulla is not only traversed by many of the most important afferent and efferent pathways of the nervous system, but is the site of decussation of several systems of fibers, notably the mesial fillet, Deitero-spinal, olivo-cerebellar and pyramidal tracts.

AN ATYPICAL CASE OF PROGRESSIVE MUSCULAR DYSTROPHY

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The distinguishing characteristics of progressive muscular dystrophy as opposed to progressive spinal muscular atrophy are given by Oppenheim as follows: "1. Onset at an early age. 2. Its hereditary or familial occurrence in most instances. 3. The commencement of the dystrophy * * * usually in the pelvic girdle and the muscles of the vertebral column, of the thigh or of the shoulder girdle and upper arm. 4. The combination of the atrophy and pseudo-hypertrophy." He also makes three subgroups, which he designates as follows: the "infantile" or Duchenne-Landouzy-Dejerine type, in which there is a primary involvement of the facial muscles; the "hereditary" type, in which the muscles do not show the pseudo-hypertrophy; and the "Leyden-Mobius" type, showing the changes mainly in the lower extremities.

The case to be presented is of interest for the following reasons: the onset at the age of sixteen (although there is a possibility that there was facial involvement earlier); the absence of heredity; the first recognized appearance in the muscles of the shoulder girdle, and the absence of any appreciable degree of pseudo-hypertrophy. The case presents some of the symptoms of all of the three subgroups mentioned previously.

HISTORY

The case is that of R. P., No. 27230, a female, single, twenty years of age, of Italian descent, who has housework for an occupation.

The family history reveals no evidence of consanguinity, nervous or mental diseases.

The patient is the youngest of a fraternity of seven children. Two died in infancy of cholera infantum. The others, aside from the patient, have arrived at maturity and are considered normal, mentally and physically.

The gestation and birth of the patient were normal. She was a healthy child and there is only the history of an attack of measles during childhood. Her health was considered good up to the advent of puberty at the age of thirteen. From that time she did not seem so strong, although no history of any definite illness can be obtained except that her menstrual periods, which were profuse and lasted five days, probably had a debilitating effect on her. She began school at the age of six years and finished two years of high school by the age of seventeen. The onset of the present trouble was the determining factor in leaving school at this time. The patient has used wine moderately. She is considered by the other members of the family rather excitable, and lately more irritable.

The disability which brought the patient to this Clinic for examination and advice had its onset about five years ago, at the age of fifteen. The first symptom noticed was that it required more of an effort than usual for her to raise her right arm to her head in combing her hair. She soon noticed that the left arm was similarly affected and it was not long before it was impossible for her to perform this act without resting her elbows on the dresser. Some time later a member of the family noticed that the patient's scapulae were becoming more prominent and movable. The patient soon noticed a sense of fatigue in the back, hips and legs when walking short distances. This weakness has necessitated her sitting down to rest frequently when walking on the street. She has some difficulty in climbing stairs, as she cannot dorsi-flex the feet well, and for this reason if given a sudden push from the rear will fall, as she cannot maintain her equilibrium. This disability has limited her activities not a little and has precluded dancing. The reported increased irritability can

be accounted for largely as the natural reaction of a girl of unusually vivacious temperament, whose means of expression of this vivacity have been markedly curtailed.

EXAMINATION

The examination showed the following: A well-nourished adult with arms and lower extremities proportionately better developed, apparently, than the trunk, although the trunk did not seem to resemble the "wasp waist" described by Marie.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—When the arms are raised from the sides, the superior angles of the scapulae are rotated upward and outward so that the prominence made by them is visible anteriorly, external to the root of the neck. When viewed posteriorly the vertebral borders of the scapulae are so far removed from the posterior chest wall that it is possible to grasp them between the thumb and fingers. The mouth is pouting and of the "tapir" type.

Gait.—When the patient walks there is an abnormal mobility of the pelvis. The gait is rather slow and deliberate, as the patient is afraid of falling. Walking on the toes is unimpaired, but walking on the heels is impossible due to weakness of dorsi-flexion of both feet. She had no difficulty in walking a line, walking backwards, or sideways, except that she showed the same caution mentioned previously.

Coordination.—The patient is right-handed. There was no equilibratory or non-equilibratory ataxia shown, as she was able to stand with feet together, eyes open or closed, without swaying; stood on each foot separately and did finger-to-nose and finger-to-finger tests without difficulty. The toe-to-object and heel-to-knee tests in the recumbent position were well executed. Adiadochokinesia and dysmetria were not present.

Skilled Acts.—Dyspraxia, apraxia, abnormality of handwriting, or speech defects were not noted.

Abnormal Involuntary Movements.—

There was a fine tremor of the extended hands noted. No fibrillary twitchings, choreiform movements, athetosis or spasms have been in evidence at any time.

Reflexes.—The jaw, biceps, triceps, ulnar and wrist reflexes were active. The radial, suprapatellar, patellar and Achilles reflexes were present, active, and apparently equal on the two sides; the tibio-adductor, direct or crossed, were not obtained. The abdominal reflexes were present and normal, although the right lower may have been a little less active than the left. The same inequality was noted in the plantar reflexes, except that here the right was the more active. Babinski, Oppenheim, Gordon, Chaddock, and Schaefer's reflexes were not elicited.

Muscle Strength.—The patient could not flex the trunk on the thighs when in a supine position. When arising from a supine position she did so as follows: she placed the left hand palm down on the bed under the lumbar region of her back, and pushed the trunk into an upright position. She then curled the feet under her, and with left hand on the bed, the right hand on the thigh, she pushed the body forward and upward until the trunk rested on her heels, and then with a vigorous push with both hands and a spring, she arose to an erect position. It was practically impossible for her to do this slowly, especially the last movement. When arising from a sitting posture to the erect position she propelled the body forward off from the chair, the thighs maintaining a horizontal position, until the trunk was over the heels, and then with both hands pushing on the thighs, she sprang into the erect position. She could not perform the act slowly. The movements of the head showed that the muscles of the neck were not affected, as there was no apparent weakness in unopposed or opposed movements. There seemed to be slight weakness of the orbicularis oris muscle, giving the mouth a "tapir" appearance. The movements of the shoulders in all directions were impaired to about half of their normal efficiency, the right being a little less capable than the left. The arms could not be raised above an angle of

seventy-five degrees in any direction, and had little more than sustaining power at that point. The forearms showed a normal strength of flexion, extension, pronation and supination, except in the latter movement, where the right was a little less competent than the left. The trunk could not be raised to the erect position if it was flexed or extended over an angle of forty-five degrees from the vertical position. The thighs could be flexed on the trunk, but had little more than sustaining power for longer than a second. The same weakness was shown in extension, adduction, abduction, inward and outward rotation, although the weakness was not quite as pronounced in the last four movements as in the first two. The legs could be readily flexed on the thighs, but had little more than sustaining power against pressure; extension was normal. Dorsi-flexion of the feet had little more than sustaining power, while plantar flexion seemed to be normal. Lateral movement of the feet was well executed. The toes showed the same weakness as the feet in dorsi-flexion.

MUSCLE STATUS.—There was marked atrophy of the muscles of the shoulder girdle, particularly the serratus magnus, supra- and infraspinati and the pectorali major. The thighs and calf groups were of good size, but did not have the characteristic feel of either hypertrophy or pseudohypertrophy.

ELECTRICAL REACTIONS.—The electrical reaction of the muscles of the right shoulder girdle were hyperexcitable to F and G as compared with the same muscles of the left side. The thigh and leg groups were hypoexcitable to F and G. RD was nowhere present.

ABNORMAL ASSOCIATED MOVEMENTS.—Grasset-Bychowski's sign (inability to hold up both legs) was present.

GENERAL SENSORY.—Touch, including discrimination, was normal except that in

the toes two points were from five to seven millimeters apart. Pain, deep and superficial, temperature, vibratory sense, muscle tendon sense and stereognosis were not disturbed.

CRANIAL NERVES.—Showed no deviation from the normal.

MENTAL STATUS.—No abnormalities were elicited, except possibly the reported increased irritability, which seemed to be the reaction natural to an altered mode of life and the foregoing of many of the pleasures which contribute to the life of a normal person of the age of the patient.

SYSTEMIC.—The thyroid showed some enlargement in the middle lobe.

Blood pressure: Systolic—98, Diastolic—70.

Wassermann in the blood—Negative.

SUMMARY

The case presented is that of a young adult with negative family history and a normal birth, childhood, and adolescence. About the time of puberty she showed a general lessening of physical strength, and at the age of fifteen, or about five years ago, definite muscular weakness appeared in the right upper extremity, then the left, and consecutively the back, hips, and lower extremities. Shortly after the muscular weakness made its appearance, atrophies of the muscles of the shoulder girdle were noted, and these have been progressive, until a typical "winged scapula" is present on each side. A resumé of the muscular condition is as follows: a slight weakness of the orbicularis oris giving a "tapir" mouth effect; weakness of all the muscles of the shoulder girdle to about one-half of their normal efficiency; preservation of the muscular strength of the forearms and hands; a weakness of the anterior and posterior trunk groups, more especially the latter; weakness of the posterior thigh group and the anterior leg group, including the dorsiflexors of the feet and toes; no atrophies below the waist

line, but possibly some hypertrophy or pseudo-hypertrophy of the thigh and calf groups. The mode of arising from a supine to an erect position and from a sitting to an erect position is particularly characteristic. The electrical reactions show only a quantitative change in excitability and in no instance was there an alteration of polarity.

The onset, course, subjective and objective symptoms exhibited, including the mode of arising from the supine and sitting positions to the erect position, together with the electrical reactions, seem to indicate that we are not dealing with a spinal or neural condition but with a primary myopathy of the progressive muscular dystrophy type.

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